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ARCHIVES OF SURGERY

VOLUME 17 AUGUST, 1928 NUMBER 2

FAT EMBOLISM

II. INCIDENCE AT POSTMORTEM *

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In an earlier report on uncompleted studies on fat embolism,[1] the conclusion was reached that fat embolism is not necessarily traumatic in origin. This conclusion was based partly on experimental work and partly on published reports of the occurrence of fat embolism in nontraumatic cases. At that time, our personal experience did not include any instances of the latter type in the human being. Further, we found that search for fat emboli is not a routine in pathologic examination postmortem. It therefore seemed desirable to check earlier authors by a search through unselected postmortem material for cases of fat embolism.

In our previous observations, the question was also raised as to the problematic rôle of fat embolism in the production of symptoms and death even in extremely severe cases. We found that dogs would tolerate without symptoms an amount of free fat in the blood stream considerably greater, weight for weight, than the amount that could be freed in the human being by the ordinary fracture, even of the femur. Our conclusions on this figure are somewhat modified by later injection experiments now being prepared for publication; nevertheless, we shall report a discrepancy in tolerance that still casts doubt on certain postmortem diagnoses of fat embolism as a cause of death. It was hoped that the present series of examinations of material observed at autopsy would yield information concerning the quantity, distribution and character of fat emboli in various disease states that might throw some light on this second question.

In our earlier studies, we concluded that the fat found as embolism in nontraumatic cases probably appeared by the coalescence of the ultramicroscopic particles of fat normally occurring in the blood plasma. This coalescence was attributed to a breaking down of the physiologic emulsion from physical or chemical changes in the blood and was pro-

* From the Department of Surgery of Washington University and the Barnes Hospital.

1. Lehman, E. P., and Moore, R. M.: Fat Embolism: Including Experimental Production Without Trauma, Arch. Surg. **14**:621 (March) 1927.

duced experimentally in the living dog by the administration of ether. A further possible source of this fat is suggested by the present study.

The presence of fat embolism following death from causes other than trauma has been remarked by several observers. Scriba [2] found fat embolism present in 52 per cent of all bodies. In addition, many specific diseases have been found to be associated with fat embolism. To put these on record in juxtaposition to our present results, we quote the following paragraph from our previous report: [1]

> Carrara [3] reports fat embolism present at necropsy in 22 per cent of deaths from cardiovascular-renal disease and in 44 per cent of burns. Catsaras [4] found fat emboli in the lungs in eighteen of sixty-seven cases of postinfluenzal pneumonia. Gröndahl [5] reports fat embolism in two cases of eclampsia, four cases of diabetes, and two cases of phosphorus poisoning; Winkler [6] in two cases of acidosis, and Winogradow [7] as occurring regularly in potassium chlorate poisoning. In addition to these repeated findings there have been authentic reports of fat embolism having occurred, without history of trauma, in one or more cases of each of the following conditions: carbon monoxide poisoning,[3] profound sepsis,[8] chronic alcoholism,[5] chloroform narcosis,[6] diabetic retinitis,[8] phlegmonous gastritis,[5] acute pancreatitis,[3] chronic tuberculosis,[8] menstrual suppression, hepatitis, splenitis, carcinomatosis and sarcomatosis.[9] (The last five conditions are cited from the literature by Warthin.)

On account of the generally accepted observation that if fat embolism occurs, it will be seen in the lung (because of the slower blood current and unsupported capillary walls), our observations were limited to lung tissue. This limitation gave us also a simpler standard for comparison. We have examined the available lung tissue in fifty unselected cases that were subjected to autopsy by the department of pathology of Washington University. These cases were not numerically consecutive, but they were consecutive with the necessary proviso that lung tissue had been preserved and filed. They were known to us by serial number until the tissue had been sectioned, stained and diagnosed for fat embolism. Sudan III was uniformly used for staining. After our diagnoses were recorded, we referred to the files of the pathologic department for postmortem diagnoses. We thereby prevented any preconceived ideas from influencing the result.

It is to be noted that the material available consisted of only one piece of tissue in most cases. In those instances in which disease of the lung was present, the specimen usually included the diseased portion of the

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2. Scriba: Deutsche Ztschr. f. Chir. **12**:118, 1879.
3. Carrara: Freidreich's Bl. f. gerichtl. Med., 1898, vol. 49.
4. Catsaras, J.: Presse méd. **26**:618 (Sept. 4) 1920.
5. Gröndahl: Deutsche Ztschr. f. Chir. **111**:56, 1911.
6. Winkler: Ztschr. f. orthop. Chir. **45**:616, 1924.
7. Winogradow: Virchows Arch. f. path. Anat. **190**:92, 1907.
8. Bantin, C. F.: Diabetic Lipemia Retinalis and Fat Embolism, J. A. M. A. **86**:546 (Feb. 20) 1926.
9. Warthin: Internat. Clin. **4**:171, 1913.

organ. It is possible that this fact influenced the results; however, as intracapillary fat was sought, the presence of disease, such as pneumonia or tuberculosis, should tend rather to diminish the fat found through inflammatory occlusion of capillaries. If fat was found in the vessels of a small segment of one lung in nondiseased areas, the conclusion that it occurred elsewhere in the lungs is highly probable.

Intracapillary globules of fat, except as discussed later in relation to fat found in the phagocytes, were diagnosed as fat embolism. Most of these were large enough actually to plug capillaries. The number of these was estimated, and the degree of embolism classified as shown in table 1. "Slight" fat embolism (table 1) was diagnosed when considerable search through a section was necessary before fat was found. "Moderate" fat embolism (table 1) signifies that the fat was easily found, but not in the majority of microscopic fields. "Marked" fat embolism (table 1) means that fat was found in the majority of fields. In two cases, the embolism found was limited to one area of the section. These two cases were given a separate classification (table 1) on the chance that this distribution might invalidate the diagnosis. For the reasons to follow, however, it is permissible to assume other areas elsewhere in one or both lungs.

As we have observed in our experiments on injection of fat [10] and in these present specimens, even marked grades of embolism are patchy in distribution. This is shown by areas of intense distribution of fat next to areas free from fat. For this reason, the demonstration of a few definite fat emboli (as listed in table 1) must be considered positive instances of fat embolism.

We did not find fat tissue in the peribronchial zone in the human material studied as we did in the dog. This may be because a block of tissue in the former does not reach the major bronchial ramifications as a block of the same size does in the latter.

Phagocytes containing fat were rather less regularly observed in the human being than in the dog. This probably can be ascribed to the fact that death in the human being is frequently preceded by starvation as it is not in the experimental animal. We were careful, however, to assure ourselves that no fat diagnosed as emboli was in the bodies of phagocytes. Occasionally, we found collections of fat-containing leukocytes and in the same zone much intracapillary fat (fig. 1). In one instance, this was entirely in the form of minute droplets of the same size as those in the cells. This was reported as showing only leukocytic fat (table 1). In other instances, in addition to such small droplets, globules larger by several diameters than any found in the cells were present. These droplets were reported as emboli. It seems possible that a source of free fat in the blood stream may be the destruction of fat-containing leukocytes as well as the breaking down of the physiologic emulsion of

10. Lehman, E. P., and McNattin, R. F.: To be published.

TABLE 1.—*Degree of Embolism in Fifty Unselected Cases*

		Age	Operation	Trauma
	NO FAT EMBOLISM			
	No Fat			
2901	Prematurity	Premature	0	0
2909	Acute purulent mastoiditis; hemorrhage into brain	10 wks.	0	0
2913	Asthma, bronchial; bronchitis, acute and chronic	60	0	0
2945	Congenital atresia, common hepatic duct	6 wks.	0	0
2962	Enterocolitis, acute; bronchopneumonia	4½ mos.	0	0
2968	Tumor of cerebellum	29	{ 7 mos. ago 24 hrs. ago	0
2984	Congenital heart disease	6 mos.	0	0
2986	Coronary thrombosis; rupture of heart	50	0	0
3006	General purulent peritonitis following perforation of gastric ulcer	49	5 days ago	0
3008	Carcinoma of head of pancreas, metastasis to liver	55	0	0
3315	Miliary tuberculosis (general) 3 weeks, postpartum	25	0	3 wks. (labor)
	Leukocytic Fat Only			
2949	Acute bacterial endocarditis, four transfusions	28	0	0
	Fat in Alveolar Exudate Only			
2947	Pulmonary tuberculosis; tuberculous pneumonia	24	0	0
	FAT EMBOLISM			
	Slight			
2905	Lobar pneumonia; chronic tuberculosis	52	0	0
2910	Acute lymphatic leukemia	2	0	0
2916	Excision of cecum; acute suppurative peritonitis	6	Recent	0
2922	Endocarditis, chronic and acute; pulmonary thrombosis	44	0	0
2925	Glioma of brain	30	Recent	0
2931	Glioma of brain; hemorrhage, postoperative	5	Recent	0
2936	Cholecystitis and cholelithiasis; general peritonitis; abscesses of liver	50	6 days ago	0
2938	Acute purulent otitis media and mastoiditis, bilateral	2 mos.	0	0
2941	Endocarditis, chronic	39	0	0
2946	Acute lobar pneumonia	45	0	0
2950	Carcinoma of stomach, with metastases	43	0	0
2951	Hypertrophy and dilatation of heart; endocarditis, chronic	40	0	0
2955	Abscess of lung; pyemia	?	0	0
2958	Adenoma of thyroid; auricular thrombus	59	0	0
2962	Polycystic kidney, bilateral	42	Recent	0
2964	Cellulitis of thigh, with abscess; bronchopneumonia	46	2 days ago	0
2965	Aneurysm of arch of aorta (wiring operation)	51	1 year ago	0
2989	Lobar pneumonia; empyema, thoracic	33	0	0
2994	Status lymphaticus; old operative scar for nephrostomy	1½	Old	0
3010	Chronic myelogenous leukemia; bronchopneumonia	26	0	0
3011	Toxemia of pregnancy; incision, peritonsillar abscess	36	Recent	0
3020	Syphilis, congenital	4 mos.	0	0
	Moderate			
2883	Carcinoma of cervix; acute pelvic peritonitis	42	0	0
2895	Carcinoma of kidney; pyonephrosis	52	0	0
2914	Toxemia of pregnancy; retained placenta	21	0	Recent (labor)
2930	Abscess, subdiaphragmatic; cholecystectomy	50	Recent	0
2957	Mastoiditis, acute, bilateral; paracentesis tympani	15 days	Recent (see col. 2)	0
2961	Fracture, ribs, multiple; emphysema, subcutaneous	86	0	Recent
2975	Arterial sclerosis, involving coronary artery	58	0	0
2996	Hypertrophy of prostate; pyelonephritis; pericarditis, adhesive	49	0	0
3005	Chronic ulcerative colitis with polyposis; acute purulent peritonitis; ileostomy	54	Recent	0
3017	Aneurysm of abdominal aorta with rupture; syphilis	62	0	0
	Marked			
2933	Subperiosteal abscess, femur; suppurative arthritis, hip; pyemia	7	0	0
2935	Adenocarcinoma, colon, with metastases; spontaneous fistula	57	8 days ago	0
2960	Arthritis, knee, suppurative; thrombosis of coronary artery; incision and drainage, knee joint	63	Day of death	0
	Embolism in Single Area of Section			
2906	Lymphatic leukemia	52	0	0
2923	Glioma of pons; bronchopneumonia	35	Recent	0

the plasma in nontraumatic cases. This is borne out by the fact that, with the one exception noted, whenever leukocytes were filled with fat, embolism also occurred.

The statistical analysis of the results of this study is reported in tables 2, 3 and 4. Of course, in such a small series, no emphasis can be put on the actual percentage estimations as indicating definite rates of incidence. They serve, however, to make certain conclusions tenable. The striking facts obtained from this analysis are as follows: 1. Definite fat embolism occurs in more than half of all lungs examined at

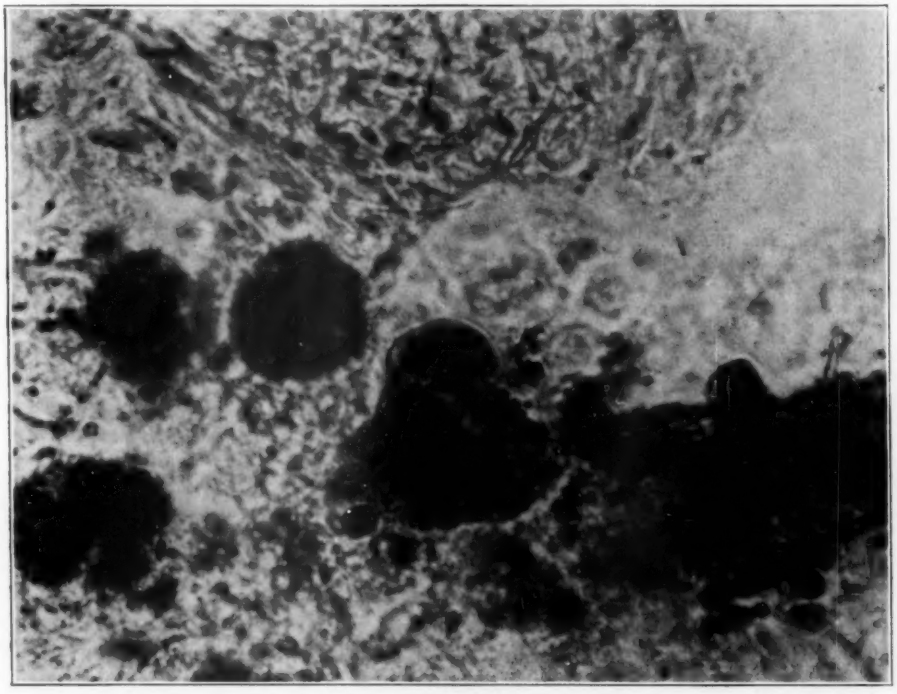

Fig. 1.—Fat emboli and fat-filled phagocytes; showing the difference in size between globules of free fat diagnosed as emboli and the much more minute particles in the cells (autopsy no. 2935); × 986.

autopsy (table 2). 2. About one half of the lungs in which fat embolism occurs are from patients in whom there has been no history of trauma (table 3). 3. In a small proportion of cases in which definite trauma is recorded, no emboli could be found in a single examination of one lung (table 3). 4. Fat embolism is found in an overwhelming proportion of lungs from patients subjected to injury or operation (table 4). 5. Fat embolism occurs in more than half of the miscellaneous fatal diseases without history of trauma (table 4).

The results of this study confirm earlier conclusions that fat embolism is an extremely common postmortem observation in all kinds of conditions, traumatic and nontraumatic. If the proportion found in this series of fifty cases should hold true through a larger experience, more than

TABLE 2.—*Proportionate Division of Fat Embolism in Fifty Unselected Lungs*

				Per Cent
No embolism			13	26
Embolism			37	74
Slight	22	44%		
Moderate	10	20%		
Marked	3	6%		
Single area	2	4%		
Total			50	

TABLE 3.—*Relationship of Trauma to Occurrence of Fat Embolism in Fifty Unselected Lungs*

		Per Cent
No Fat Embolism		
Traumatic	3	23
Nontraumatic	10	77
	13	
Fat Embolism		
Slight		
Traumatic	9	41
Nontraumatic	13	59
Moderate		
Traumatic	5	50
Nontraumatic	5	50
Marked		
Traumatic	2	66.6
Nontraumatic	1	33.3
Single areas		
Traumatic	1	50
Nontraumatic	1	50
Summary		
Traumatic	17	46
Nontraumatic	20	54
	37	

TABLE 4.—*Occurrence of Fat Embolism in Traumatic and Nontraumatic Cases in Fifty Unselected Lungs*

		Per Cent
Traumatic cases	20	
No embolism	3	15
Embolism	17	85
Nontraumatic cases	30	
No embolism	10	33.3
Embolism	20	66.6

half of all fat embolism is not associated with trauma. The variety of diagnoses in nontraumatic cases in which we have found fat embolism is great. Certainly no one influence can be detected. Although, in our tables, the assumption is made that trauma is the determining cause of fat embolism in those cases in which it occurs in the history, the frequent

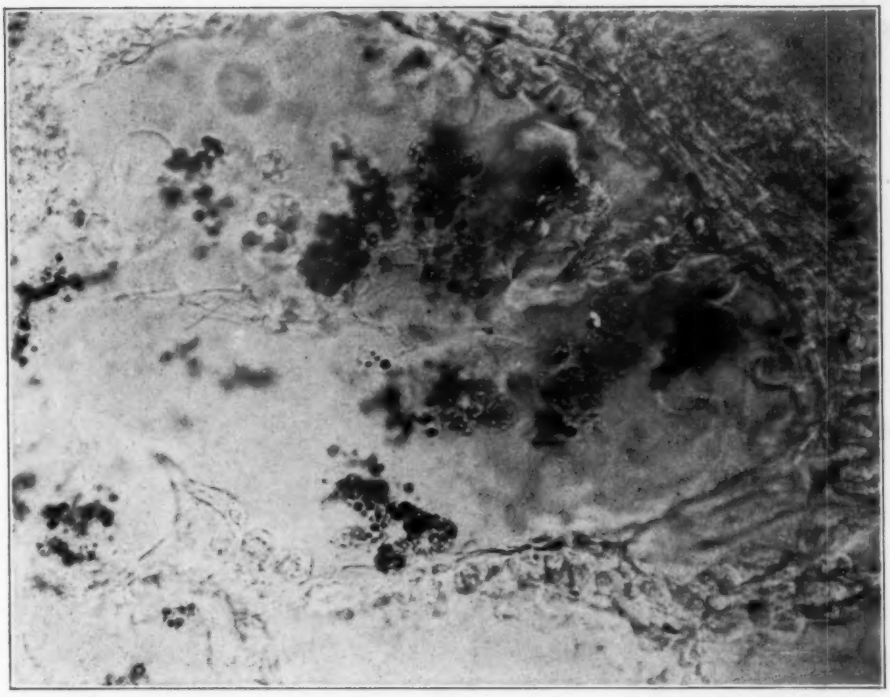

Fig. 2.—From a case of tuberculous pneumonia; showing free fat and fat-filled cells in the alveolar exudate and no fat in the alveolar walls (autopsy no. 2947). This illustrates a possible fallacy in Warthin's sign; × 490.

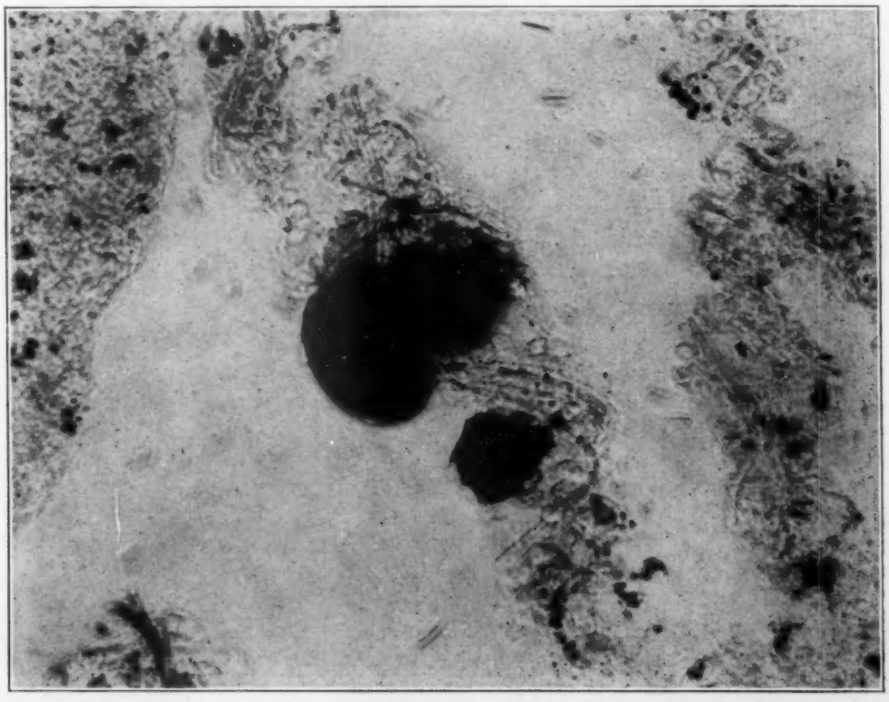

Fig. 3.—Fat emboli following multiple fractures of the rib (autopsy no. 2961). Here a globule of fat appears to be partially extruded into the adjacent alveolus, illustrating the mechanism of the appearance of fat in the sputum (Warthin's sign of fat embolism); × 490.

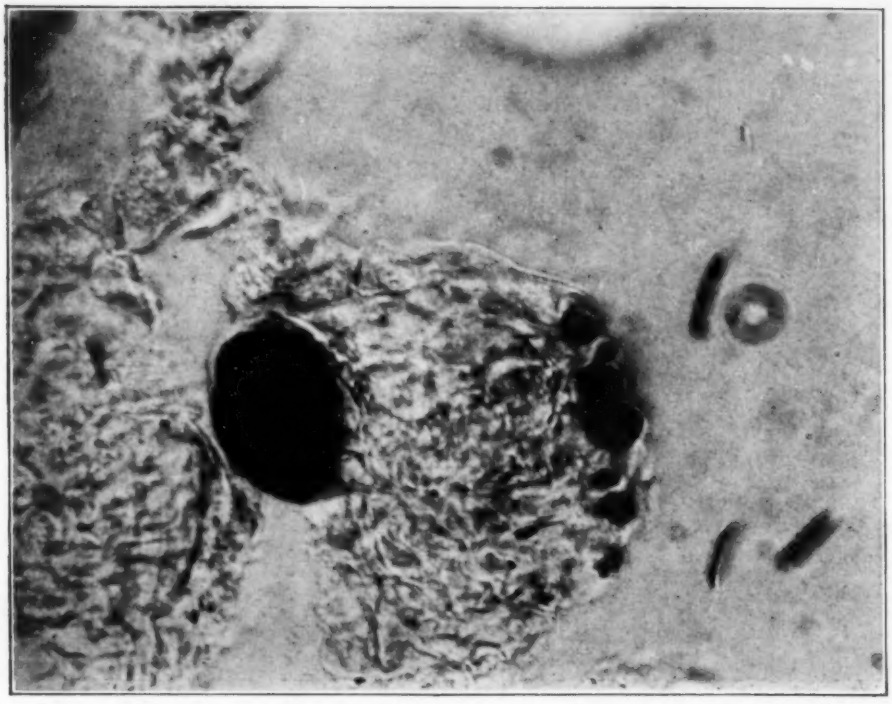

Fig. 4.—Fat emboli in a case of toxemia of pregnancy with retained placenta (autopsy no. 2914); × 986.

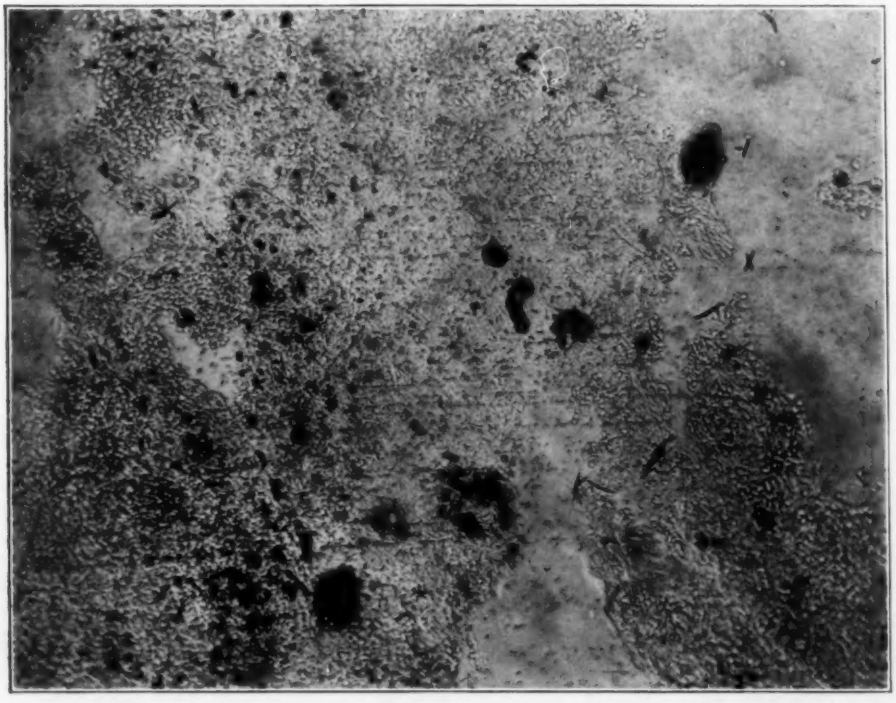

Fig. 5.—Fat emboli in case of suppurative arthritis of hip with subperiosteal abscess of femur in which operation was not performed (autopsy no. 2933). Low power magnification was used to show large number of emboli.

occurrence of fat embolism in nontraumatic fatal disease makes such an assumption not necessarily true. In other words, in some of the cases reported as due to trauma, other factors may conceivably have been the true cause.

What these factors are is a pressing problem. As reported earlier, there is certain experimental evidence that they may consist in physical and chemical changes in the blood, converting the physiologic emulsion of the plasma into free fat. This has been accomplished in the dog by the administration of ether. In studies of artificial emulsions in the test tube,

Fig. 6.—Fat emboli in case of suppurative arthritis of knee with coronary thrombosis. This patient was subjected to incision and drainage of the knee joint on the day of death (autopsy no. 2960); × 490.

there is suggestive evidence that the products of protein decomposition may possibly be blamed. Further studies along these lines have so far not been successful in confirming this suggestion.

When one considers these facts together with the experiments already referred to, one is forced to the conclusion that the presence of fat embolism in the lung after trauma does not justify the conclusion that such fat has been caused by the trauma or that it has caused death. Unquestionably there are cases of multiple fractures showing immediate symptoms, early death and blood at postmortem loaded with fat,

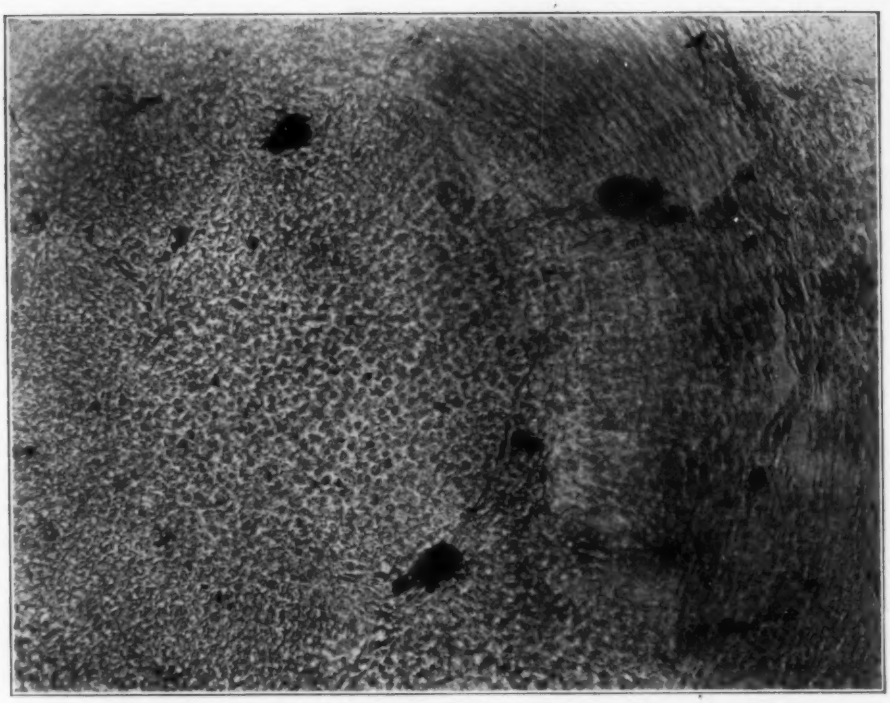

Fig. 7.—Slight degree of fat embolism (autopsy no. 2905). Postmortem diagnosis was lobar pneumonia and chronic tuberculosis. Three emboli were found in a single field; × 183.

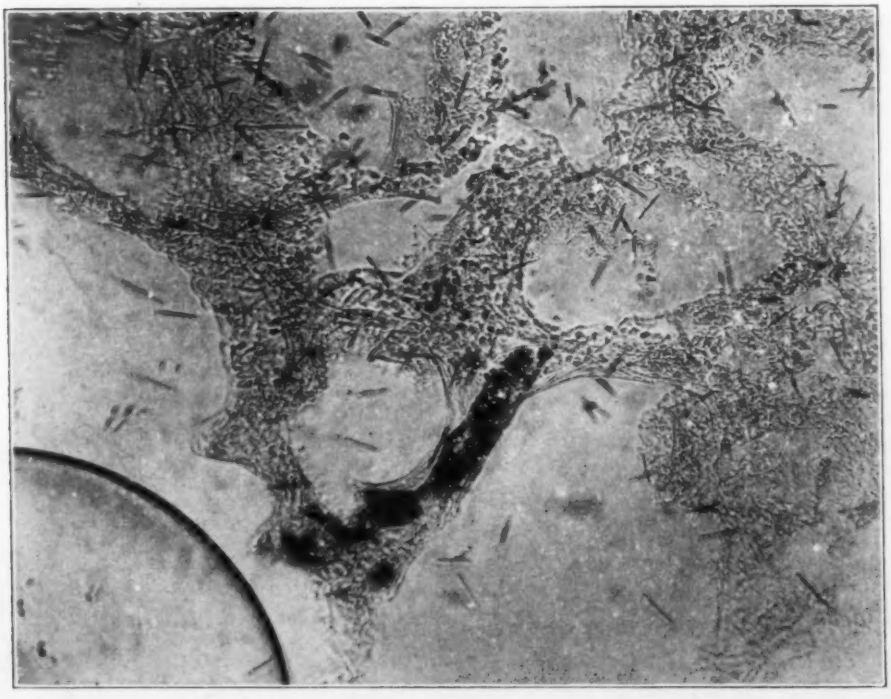

Fig. 8.—Several fat emboli in a case of carcinoma of the kidney and pyonephrosis (autopsy no. 2895); × 198. Precipitated crystals of Sudan III are seen in this and other photomicrographs.

in which so far as we know at present, it must be considered that the patients died of fat embolism. On the other hand, in any case of trauma with delayed symptoms, no matter how rich the blood in free fat, one must question the direct relationship of the trauma to the fat embolism and the relationship of the fat embolism to the symptoms. It is at least conceivable that the influences causing symptoms and death are also the influences causing freeing of fat in the blood stream.

There is one interesting note to be added. In a case of tuberculous pneumonia observed at autopsy (table 1, no. 2947), a large amount of fat was found in the alveolar exudate and none in the blood vessels (fig. 2). It is, of course, common to observe fat in the alveoli when fat embolism is present (fig. 3). On this basis, Warthin's sign of fat embolism was devised, namely the presence of fat in the sputum. The present observation demonstrates that fat can occur in the air spaces of the lung without embolism; in other words, Warthin's sign may not be as clearcut as is generally thought.

ARTERIOVENOUS ANEURYSM OF THE BRAIN*

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The intracranial arteriovenous aneurysms here described are not peculiar to the brain. With the exception of differences of detail due to environment, they are essentially the same as arteriovenous aneurysms situated elsewhere in the vascular system.

The discovery of an arteriovenous aneurysm is always credited to William Hunter.[1] Enthusiastic phlebotomists of that period prepared two perfect examples of arteriovenous aneurysm for Hunter, which he was quick to recognize. His description of the signs and symptoms left little to be added, even at the present time. At the point of the communication between the artery and the vein, he recognized a loud hissing murmur and a strong tremulous thrill; large tortuous venous sacs were seen to pulsate; the brachial artery was greatly enlarged and serpentine cephalad to the arteriovenous fistula, but distal to it, the artery became smaller than on the other side. He was able to reduce the size of the veins, stop their pulsation and eliminate both the murmur and the thrill by pressing on a localized spot, which he recognized to be the opening between the artery and the vein. It was Hunter who first suggested the term "anastomosis" to denote the union of the two vessels.

To arteriovenous aneurysms of traumatic origin have since been added a great group with precisely the same clinical features, but in which the characteristic vascular changes have been present since birth. Virchow [2] referred to cases of Krause (Wm.) and Breschet as demonstrating beyond doubt the congenital origin of such cases. He also mentioned a case which Langier watched during the patient's life and in which he performed dissection after the patient's death; a free communication was found between the posterior auricular artery and a nearby vein. There are now many cases in which dissections of these aneurysms at operation or necropsy have demonstrated communication between a large artery and a contiguous vein by one or more aberrant vessels which could have arisen only as errors of vascular development in the embryo. In recent years, remarkable examples of these anomalous congenital communications in the neck and extremities, as disclosed at operation, have been reported by Halsted,[3] Rienhoff [4] and Reid.[5]

* From The Johns Hopkins Hospital and University.

1. Hunter, W., cited by Callender: Bull. Johns Hopkins Rep. **19**:260, 1920.
2. Virchow: Die Krankhaften Geschwülste, Berlin, 1863, vol. 3.
3. Halsted, W. S.: Congenital Arteriovenous and Lymphaticovenous Fistulae, Tr. Am. Surg. Ass., 1919.
4. Rienhoff, Jr., W. F.: Congenital Arteriovenous Fistula, Bull. Johns Hopkins Hosp. **35**:271, 1924.
5. Reid, M. R.: The Effect of Arteriovenous Fistula upon the Heart and Bloodvessels, Bull. Johns Hopkins Hosp. **31**:43, 1920.

Finally, there is a third group of cases in which an arteriovenous communication is established through the medium of a mass of abnormal vessels which take the place of the usual capillary bed, erroneously known as angiomas because of the impression that they are new growths; the true character of these lesions has long been obscured. The component vessels may be atypically arterial or venous, or both may be combined. Virchow discussed these tumors at length, and with some hesitation classified them under the heading "angioma racemosum arteriale." The following excerpt from his "Krankhaften Geschwülste" shows his grasp of the real underlying pathologic process:

> Finally there remains that form of angioma, in which the vascular enlargement is outspoken and in which the character of the tumor is more in the background. Many of these forms are not to be understood in the light of oncology. They are often classified with angiomata and one cannot make a true line of demarcation between them and angiomata. Their difference lies principally in the fact that the process is diffuse, and that dilatation of both arteries and veins is present. These tumors have recently been called by John Bell aneurysma per anastomosin, and by Walker perhaps better, aneurysma anastomoseon.

Another group of vascular tumors so well known, even at the present time, as angioma cavernosum, were included by Bell under his aneurysms per anastomosin. Virchow referred to Dupuytren's experiments to prove that in these tumors injections of colored substance passed freely from the artery into the veins through the intermediary tumor. Virchow, though not conceding the arteriovenous communication, noted the fact that in some cases of cavernous angiomas (venous tumors) the artery is dilated and tortuous just as in true arteriovenous aneurysms.

It was Virchow's view that these arteriovenous aneurysms (or angiomas) were not new growths in the usual sense, but were of congenital derivation. Subsequent embryologic and pathologic researches demonstrated that no other explanation is tenable.

Professor Halsted,[3] whose interest in this subject extended over a period of several years, and who stimulated the splendid researches and publications of Callender,[6] Reid,[5] Holman[7] and Rienhoff,[4] approached the origin of arteriovenous aneurysms from the vascular anlage in the early embryo. It is known that the adult system of arteries and veins is derived from a general capillary network; that both arteries and veins form from this capillary network; that the final arterial and venous systems are derived by extensive evolution of a vastly different primitive system. This metamorphosis is accomplished by shifting, consolidating

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6. Callender, C. L.: Study of Arteriovenous Fistula with an Analysis of Four Hundred and Forty-Seven Cases, Johns Hopkins Hosp. Rep. **19**:260, 1920.

7. Holman, E. F.: Physiology of an Arteriovenous Fistula, Arch. Surg. **7**:64 (July) 1923.

and eliminating not only capillaries but even the great venous and arterial trunks of the early embryo. It is evident that the retention of the original vascular connections between arteries and veins will readily explain the origin of the adult arteriovenous aneurysms. Dr. Halsted had in mind the arteriovenous aneurysm with aberrant congenital connecting channels. But it would seem that the maldevelopment of the original vascular bed would similarly explain the nests of abnormal vessels which are interposed between the arteries and veins and which replace the normal capillary bed.

Arteriovenous fistulas of traumatic origin are practically impossible in the brain, because the principal arteries and veins are not in juxtaposition as are the vessels of the head, neck and extremities. Arteriovenous aneurysms between the internal carotid artery and the cavernous sinus are exceedingly common, but as they are really outside the cranial chambers, and as they are in point of origin rather laws unto themselves, they need not be considered here. They are dependent on an anatomic arrangement which is not duplicated anywhere in the body. As the internal carotid arteries traverse the cavernous sinus in their passage through the base of the skull, a traumatic rupture of one of the arteries by a fracture of the base of the skull must inevitably result in an arteriovenous fistula, unless or until the arterial opening is repaired. The nomenclature of lesions which should really be included under arteriovenous aneurysms is most confusing. Being so dependent on the personal equation for the interpretation, they are reported under many titles, i.e., angioma cavernosum, varix aneurysmaticus, angioma arteriale racemosum, angioma plexiforme, aneurysma cirsoïdea, aneurysma serpentina, aneurysma anastomotica, arteriovenous aneurysm, tumor cirsoïdius, and particularly by the Germans as Rankenangiom—a term first applied by Virchow. Rienhoff [4] suggested a simple and sensible revision of the nomenclature of all vascular dilatation and communications into (1) venous, (2) arterial and (3) arteriovenous aneurysms, thereby discarding the term hemangioma at least for major lesions. This classification meets my full approval. In the group of arteriovenous aneurysms, the various terms "cavernous," "racemose," "cirsoid" and "serpentine," whether applied to arteries or veins, are merely descriptive of a superficial expression of a lesion and not of the fundamental pathologic process. A long as the term hemangioma is employed when an arteriovenous connection exists, there must always be uncertainty as to whether the tumor is arterial or venous. As a matter of fact, they may be either or both, but regardless of these superficial differences there is a direct communication through the vascular abnormality which has replaced the normal capillary bed. Moreover, the outward expression of an arteriovenous aneurysm, i.e., the arterial tortuosity and dilatation and the

engorgement of the veins leaving the tumor, are doubtless identical whether the communicating channels are exclusively arterial or venous or predominantly one or the other.

In attempting to gather the cases of arteriovenous aneurysms of the brain, the records of venous aneurysms and the various subdivisions of angiomas have been reviewed, and most of them have been collected from these groups. For example, there is no more perfect example of an arteriovenous aneurysm than the one pictured in Krause's colored plate,[8] but which he calls a venous angioma. On the other hand, cases reported as Rankenangioma and cirsoid aneurysma, and therefore presumably arteriovenous aneurysms, have been excluded from this group because the absence of changes in the arteries and veins leading to and from the tumor has suggested a pure venous lesion. Other cases reported as venous, which undoubtedly seem to be arteriovenous, are those of Astwazaturoff,[9] Steinheil,[10] Dürck, Kalischer,[11] Mühsam,[12] Eiselberg,[13] Campbell and Ballance [14] and others. Doubtless the teaching of Oppenheim and Krause was in large part responsible for the interpretation of these vascular lesions as venous.

FREQUENCY OF ARTERIOVENOUS ANEURYSMS OF THE BRAIN

The first arteriovenous aneurysm of the brain was reported by Steinheil [10] in 1895, or 138 years after William Hunter's [1] first reported arteriovenous aneurysms in the arm.

A total of twenty-two cases which seemed to represent this type of intracranial aneurysm have been collected from the available records. Two additional cases of Struppler [15] and Simmonds [16] (second case) have

8. Krause, F.: Chirurgie des Gehirns und Rückenmarks, vol. 1, p. 88, 1908, English trans., New York, Rebman Company.

9. Astwazaturoff, M.: Ueber die kavernose Blutgeschwulst des Gehirns (zur Kasuistik der Pseudomeningitis), Frankfurt. Ztschr. f. Path., 1911, vol. 4.

10. Steinheil, S. O.: Ueber einen Fall von Varix aneurysmaticus im Bereit der Gehirngefoesse, Inaug. Diss., Würzburg, 1895.

11. Kalischer, S.: Demonstration des Gehirns eines Kindes mit Teleangiektasien der linksseitigen Gesichtskopfhaut und Hirnoberfläche, Berl. klin. Wchnschr. **34**:1059, 1897.

12. Mühsam, R.: Ueber Varicen und Angiome des Zentralnervensystems, Arch. f. klin. Chir. **130**:522, 1924.

13. v. Eiselsberg, A. F., and Ranzi, E.: Ueber die chirurgische Behandlung der Hirn- und Rückenmarkstumoren. Ligatur von Angiomen, Arch. f. klin. Chir. **102**:341, 1913.

14. Campbell, H., and Ballance, C.: Case of Venous Angioma of Cerebral Cortex, Lancet **1**:10, 1922.

15. Struppler: Ueber das Cavernöse Angioma des Grosshirns, München. med. Wchnschr. **37**:1269, 1900.

16. Simmonds, M.: Ueber das Angioma racemosum serpentinum des Gehirns, Virchows Arch. f. path. Anat. **180**:280, 1905.

been included, though the status of both is doubtful because the description of the venous outlets is inadequate. Several cases heretofore classified as cases of angioma, and usually assumed to be similar to those collected here, have been excluded. Among them are the cases of Oppenheim,[17] Sweasey-Powers,[18] Lewandowsky and Selberg,[19] Cassirer and Mühsam,[20] Uyematsu,[21] Creite, Abrikosoff,[22] Lechner,[23] Versé,[24] Rossolimo[25] and Bruns. It has also not been possible to include the cases of Isenschmid,[26] because neither observations made at operation nor those made at autopsy establish the clinical diagnosis. In one of his cases at least, the diagnosis is almost surely correct for, in addition to a well defined extracranial arteriovenous aneurysm, Jacksonian epileptic attacks were of long standing. The symptoms that I have considered essential for the diagnosis of an arteriovenous aneurysm have been, first and foremost, marked fulness and enlargement of the veins of exit; second, an increased size and tortuosity of the artery entering the snarled mass of vessels. Without the enlarged veins on the cortex or elsewhere one cannot assume the existence of an arteriovenous fistula.

The seeming infrequency of this lesion cannot be judged by the small number of cases assembled from the literature. Were this true, the lesion would be rare indeed. The eight cases which occurred at the Johns Hopkins Hospital among about 600 cases of verified tumors of the brain during a period of five years, are perhaps a better index of its relative frequency. In clinics on which neurologic material is concentrated, arteriovenous aneurysms apparently occur in from about 0.5 to 1 per

17. Oppenheim, H.: Die Geschwülste und die syphilitischen Erkrankungen des Gehirns, in Nothnagel's Practice, vol. 9, p. 20.

18. Sweasey-Powers, W. J.: Ein Fall von Angioma cavernosum des Gehirns, Ztschr. f. ges. Neurol. u. Psychiat. **16**:487, 1913.

19. Lewandowsky, M., and Selberg, F.: Ueber Jacksonsche Krämpfe mit tonischem Beginn und über ein kleines Angiocavernom des Gehirns, Ztschr. f. d. ges. Neurol. & Psychiat. **19**:336, 1913.

20. Cassirer and Mühsam: Berl. klin. Wchnschr. **17**:755, 1911.

21. Uyematsu, S.: A Case of Haemangioma Cavernosum of the Cerebrum, J. Nerv. & Ment. Dis. **52**:388, 1920.

22. Abrikosoff, A. S.: Ein Fall von Angiona arteriale racemosune der Arterial basilons und der beiden arterial communicans posterior, Zentralbl. f. allg. Pathol. **22**:210, 1911.

23. Lechner, Ellen: Ein Beitrag zur Kasuistik der Hirnangiome, Beitr. z. klin. Chir. **125**:174, 1922.

24. Versé: Demonstration eines in die Substanz des Kleinhirns eingebetteten Aneurysma serpentinum et sacciforme der Arteriae Cerebri posterior Sinistra, München. med. Wchnschr. **58**:544, 1911.

25. Rossolimo, G. S.: Zum Ausgang von Gehirnoperationen, Deutsche Ztschr. f. Nervenh., vol. 6; Neurol. Zentralbl. **15**:714, 1896.

26. Isenschmid, R.: Die klinische Symptome des cerebralen Rankenangioms, München. med. Wchnschr. **59**:243, 1912.

cent of the cases. It is clear that more cases will be disclosed at operation than at necropsy, because the symptoms (usually epilepsy) are not such as to require prolonged hospitalization. Most of the necropsies were obtained on patients who were brought to the hospital in coma resulting from cerebral hemorrhage. This number obviously represents but a small proportion of the afflicted persons.

CASES OF INTRACRANIAL ARTERIOVENOUS ANEURYSM

CASE 1.—*History.*—A well nourished, well built, muscular man, aged 52, an army officer, was suddenly and without warning seized with a severe clonic con-

Fig. 1 (case 1).—Arteriovenous aneurysm showing a large sylvian vein emptying into the large anomalous vein crossing the temporal and frontal lobes. There is a distinct enlargement at the point of junction of these two veins; the rolandic vein is easily defined.

vulsion lasting about fifteen minutes. This attack occurred six years prior to examination, just after his return from France, where he actively participated in the World War. He had never had any kind of an attack before. For the next three years similar attacks occurred about once every two months; then for two years there was no attack, but for the past year they have appeared about every month. Recently, there have been petit mal attacks with loss of consciousness for from forty to sixty seconds, but no muscular contractions. One week ago he had a violent generalized headache; following this, a pain persisted in the right frontal region. His head turned to the left during the convulsions, but there was no other focal sign. At times he had had a dull aching pain in the fingers of the left hand

and had complained of some stiffness in this arm. The attacks usually occurred in the early morning. He has had some trouble with his memory; he said that he often forgot most important things.

Examination.—A tenderness to deep pressure was revealed over the right parietal region. The patient said that there was a localized sore spot in this region (not over the tumor as later discovered). The neurologic examination did not reveal anything. The eyegrounds were normal. The roentgenogram of the head was negative. The Wassermann reaction of the blood was negative.

Fig. 2 (case 1).—An anomalous branch connecting the middle cerebral artery with the angiomatous mass. The transition from the artery to the vein is shown in the net work.

While he was in the hospital, an attack was observed by the nurse, but focal signs other than turning the head to the left were not seen. Urine was lost. Motor weakness did not follow the attack.

The injection of air into the cerebral ventricles showed dislocation of the ventricular system to the left side. The third ventricle was also bent obliquely to the left. The anterior horn of the right ventricle was smaller than the left. A diagnosis of right frontal tumor was made.

Operation.—Right craniotomy was performed. A group of tremendous engorged and tortuous veins covered the anterior half of the right temporal lobe, and to a much lesser extent the frontal lobe. They emptied into a huge sylvian

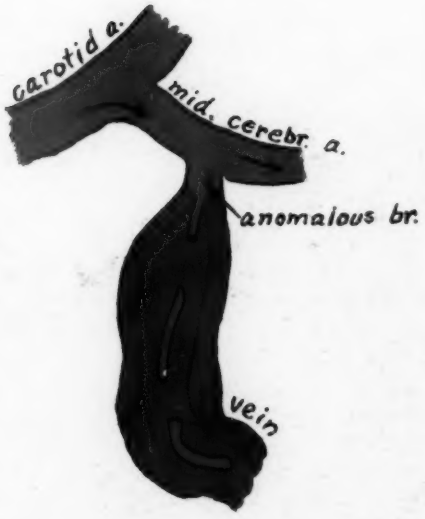

Fig. 3.—The direct communication between the artery and vein by means of an anomalous vessel.

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vein, joined at right angles a large anomalous vein which crossed the frontal lobe in a vertical direction and continued downward across the temporal lobe. There was no well defined rolandic vein. The network of veins dipped over the anterior margin of the temporal lobe, entirely obscuring the anterior pole. The largest venous trunk was over 1 cm. in diameter. The veins were a definite pink and were in striking contrast to the black veins elsewhere in the brain. One could easily see the blood rhythmically (with each heart beat) passing through the dilated thin walls of the veins. On palpation, a distinct thrill could be detected in the veins. Since any attempt to treat the aneurysm surgically appeared to involve a

Fig. 4 (case 1).—Ventriculogram showing dislocation of the ventricles to the left and the deep third ventricle. The arrows indicate the dislocation of the ventricular system toward the left.

risk greater than my co-workers and I were justified in assuming, the dura was closed and the bone flap was replaced.

Second Operation.—Two weeks later the wound was reopened, the frontal lobe retracted and the inferior surface of the frontal lobe and the anterior pole of the temporal lobe well explored. Search was at once made for an arterial communication. A large anomalous branch passed from the middle cerebral artery (immediately distal to its origin) directly into the mass of engorged serpentine vessels. It was thick-walled and definitely arterial at its origin, but less than 0.5 cm. distally there was an abrupt transition to a thin-walled vein. This vessel

was almost as large as the middle cerebral artery at this point. The arterial portion of the vessel was well isolated, surrounded by two silk sutures and ligated. The venous tumor immediately collapsed. We did not see any suggestion of a central tumor mass such as was present in other cases of the series from which the veins emerged. It was our impression that the condition was a pure arteriovenous fistula, produced by an anomalous vessel passing directly from the middle cerebral artery into a large venous trunk, which ultimately reached the sylvian vein. The great veins could be seen to connect with the sylvian vein.

Postoperative Course.—When the patient was coming out from under the effects of the ether, he complained of severe headache and weakness of the contralateral part of the arm and the leg. During the ensuing twenty-four hours, this side slowly became completely paralyzed, and his pulse rate steadily dropped to 60. Since the patient was somewhat drowsy, we suspected an extradural hemorrhage, but elevation of the bone flap did not show any collection of blood. The paralysis puzzled us, for our manipulations were entirely under the frontal lobe, too far afield from the motor tract to cause motor weakness, and seemingly remote from the middle cerebral artery. If the middle cerebral artery had by some chance been injured, why did the paralysis develop so gradually? We have also wondered whether the drop in the pulse rate might not be similar to the bradycardia noted by Holman as a constant sequel to the closure of arteriovenous fistulas in his series of dogs. Five days after the operation, recovery of function began in the left side and steadily progressed. Still unable to explain the cause of the hemiplegia, it is equally difficult to explain the recovery. Aside from a slight residual weakness of the left arm and hand and a slight limp, the patient was well three and a half years after the operation. Two and a half years after the operation, he had one convulsion; since then there have been no other seizures, either petit or grand mal, nor have there been any headaches or soreness corresponding to the old "localized tender spot." The spells of forgetfulness have disappeared.

Case 2.—*History.*—A well nourished, normal looking boy, aged 19, sought relief from convulsions (1922). At the age of 15, he was seized with numbness in the left corner of his mouth. A "pricking sensation as of pins and needles" quickly followed and spread over the left side of the face. The tongue was pulled to the left, and it also had a similar "sensation as of pins and needles." The left side of the face twitched. The sensation spread to the left arm, but the arm did not twitch. The attack lasted only about one minute. The hand was weak for several minutes after the attack; consciousness was lost. Five months later, he was attacked by a convulsion while asleep. He foamed at the mouth, and urine was passed. During the following two months there were four more such attacks, all occurring during sleep, then a free interval of several months.

One year ago, a unilateral (left side) convulsion was not accompanied by loss of consciousness, and similar attacks have since occurred about once a month, always with the warning aura of numbness at the corner of the mouth. There has been only one unconscious spell during the past year; the hand and arm are always weak after such a spell.

Examination.—The results of the examination were negative. There was no objective evidence of motor weakness on the left side. The reflexes were normal and equal on the two sides. The roentgenogram was negative. The Wassermann reaction of the blood was negative. The preoperative diagnosis was probable cerebral tumor causing jacksonian epilepsy. A tumor was suspected because motor weakness followed in the wake of the convulsion.

Operation.—Feb. 11, 1922: Right craniotomy was performed. On the dural side of the bone flap, the inner table of the skull was eroded in a circular area

about 1 by 1 cm. The roentgenogram, however, was negative. There was a corresponding localized bulge of the dura. Both abnormalities corresponded with, and were produced by, the underlying bulge in a huge vein (fig. 5). A remarkable cluster of serpentine veins was concentrated on the surface of the parietal lobe just below the arm area and extending alongside the rolandic vein and the sylvian vein. Both the rolandic and the sylvian vein were tremendously dilated; each was as large as a little finger. The central dilatation in the vein was doubtless close to the maximum point of entry of the arterial circulation. It was about 2.5 by 2.5 cm. in diameter. A cluster of smaller veins of varying size resembled a mass of angle worms. The superficial area of the group of veins was roughly circular (measuring 4.5 by 4.5 cm.) and completely obscured the underlying brain. Running anteriorly from the vascular plexus and then coursing mesially over the frontal

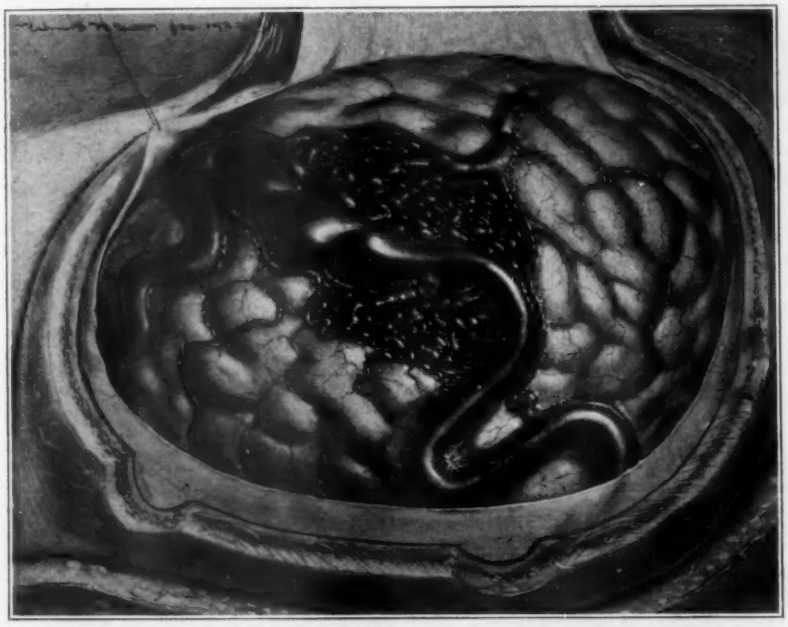

Fig. 5 (case 2).—Arteriovenous aneurysm in which the main outlet is the large tortuous rolandic vein. The cluster around the rolandic vein represents the so-called angioma. It is the connecting link between the underlying branch of the middle cerebral artery and the rolandic vein. The large vein over the frontal region is also shown and the sylvian vein which is continuous with the rolandic vein.

lobe into the superior longitudinal sinus was another abnormally large vein, though smaller than the rolandic. The main trunk of the sylvian vein which skirts the upper margin of the temporal lobe and empties into the transverse sinus was also greatly enlarged.

All of these veins pulsated like arteries. Through the thin walls of the rolandic vein the pulsing, swirling red arterial blood could be seen. The palpating finger could readily detect a thrill, not only in the large veins, but in the serpentine cluster composing the tumor. Everything indicated that the lesion was an arteriovenous aneurysm.

After consultation with the patient's brother, who realized the great danger involved, an attempt was made to extirpate the mass. It was hoped that the surface vessels might be ligated and the tumor undermined and included in a mass dissection of brain tissue, without actually coming in contact with the vessels of the tumor. As this was our first experience with this type of lesion, we little realized its extreme difficulties and dangers. The sylvian vein was tied with two silk ligatures and the vessel divided. We hoped to work through the cortex from this point mesially, but as there was not sufficient room, the line of attack was shifted to the mesial aspect of the tumor. The rolandic vein was doubly tied and divided. A remarkable resistance was encountered in ligating the vein. Usually a vein offers almost no resistance to the sliding knot, but in this instance the knot could be slipped down only step by step because of the pressure within the vein. Ligation and division of the rolandic vein were accomplished without incident. A few minutes later, however, while a subcortical dissection was being performed and preparation was being made to ligate additional vessels, a sudden burst of blood flooded the operative field. The rolandic vein had burst, not at the points of ligature but in the bulge of the vein about midway between the ligatures on its now isolated segment. The closure of the venous outlets had taken away too many of the channels of exit for blood from the tumor and the thin-walled rolandic vein, no longer able to withstand the added extra strain of increasing arterial pressure, burst at its weakest point. The bleeding became profuse. Pressure with cotton and gauze accomplished little more than to check the bleeding temporarily. A rapid extirpation of the mass was attempted, but the bleeding vessels multiplied. There was no cerebral tissue, only a skein of wormlike vessels which bled furiously. The carotid vein was tied in the neck, and while this reduced the bleeding so that tampons of cotton controlled it, shortly afterward the renewed pressure established by collateral again lifted the cotton pack, and the bleeding began anew. When it was again stopped, the patient's condition was so seriously affected that he died a few hours later.

Case 3.—*History.*—A healthy looking man, aged 35, was referred by Dr. Ralph Greene, of Jacksonville, Fla., on Jan. 25, 1925, because of a general mental and physical breakdown, headache, double vision, periods of somnolence, slurring speech and staggering gait.

The patient's condition was said to have begun two years before examination with a staggering gait and a tendency to swerve to the left. He said that he would fall to the left any time when his eyes were shut. Diplopia had been present more or less continually for the past two years. Dizziness was frequent and disturbing. A thick slurring speech had been noticed for a month. Headaches had occurred irregularly, but were more noticeable when the patient was constipated. There was also pronounced mental impairment with paranoid trend. He was irritable and suspicious and adopted a superior attitude. His insight and judgment were poor. His speech was slurring and he quickly rambled over a number of subjects without cohesion and without meaning. He had formerly studied medicine and apparently did well until he stopped because of a dislike for the subject. His memory was uncertain. Convulsions, loss of consciousness, motor or sensory disturbances or difficulty in swallowing were not noted.

Examination.—The neurologic examination showed bilateral choked disk of 3 diopters in each eye. There were no obvious extra-ocular palsies to explain diplopia; coarse nystagmoid jerks of the eyeballs occurred when the patient looked far to the right or left. There was dysarthria; greatly diminished hearing in the left ear; slight ataxia in the heel to knee test, but not in the finger to nose

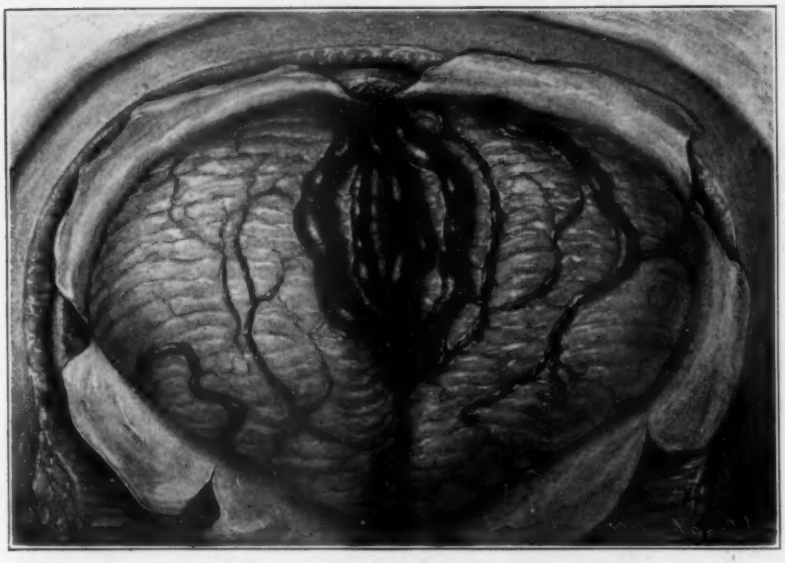

Fig. 6 (case 3).—Arteriovenous aneurysm in the posterior cranial fossa. The large veins on the surface of the cerebellum were lost under the dura.

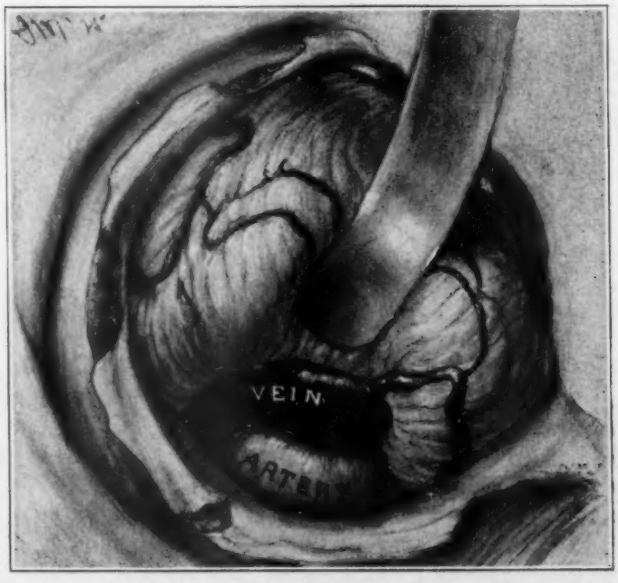

Fig. 7 (case 3).—Elevation of the left lobe of the cerebellum shows the big venous bed underlying and the tremendous vertebral artery beneath the vein.

test; staggering gait; positive Romberg sign, the patient falling constantly to the left and no adiadokokinesis. He walked with a broad base.

There were bilateral positive Babinski and Oppenheim signs; otherwise the reflexes were normal. There were no motor or sensory changes. The Wassermann test of the blood was negative. A roentgenogram of the head was negative.

A tentative diagnosis of cerebellar tumor was made, but the pronounced mental changes were unexplainable.

Ventricular estimation showed both ventricles to be large and in free communication with each other (indigo-carmine test). The fluid was under greatly increased pressure.

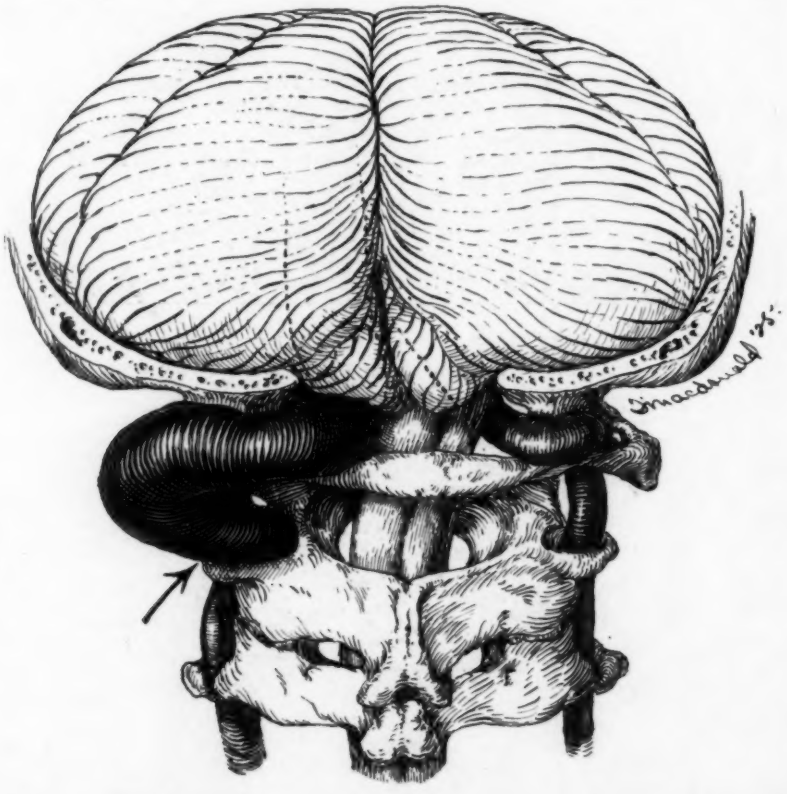

Fig. 8 (case 3).—Sketch of aneurysm showing the tremendous vertebral artery on this side. This diagrammatic sketch is intended to show the difference between the vertebral arteries on the two sides. The sketch is not accurate in that the bulge of the artery was between the atlas and the axis, but from the artist's view at the operation it might well have looked as it does. The artery was doubtless also enlarged below this point, though it could not be inspected at the operation.

Operation.—A cerebellar exploratory operation was performed. When the cerebellum was exposed, six large tortuous veins stood out in strong relief about equally on both cerebellar lobes. They converged to two large trunks at the border of the superior surface and dipped out of sight onto the tentorial surface

of the cerebellum. The danger of injury to the unusual veins made it seem inadvisable to attempt to follow the veins on the tentorial surface. Neither a thrill nor arterial pulsation was detected. Exploration of the under surface of the left cerebellar lobe brought into view two remarkable conditions: 1. The whole inferior surface of the cerebellum was covered with a venous sinus completely hiding this part of the cerebellum. 2. The vertebral artery, which usually is small and seen only after a search was much enlarged and displaced outward. It lay alongside as well as under the brain stem. Further inspection could not be made because it was fraught with too much danger. The usual bilateral cerebellar decompression was left to relieve pressure.

After the operation, the occipital region was examined with a stethoscope. A loud to and fro murmur was heard in front of and back of the ear and over the

Fig. 9 (case 3).—Patient after the vertebral artery had been ligated on the left side. The arrows are directed toward the scar after the vertebral ligation. It will be noted that the cerebellar decompression does not bulge, though it bulged markedly before this vessel was closed.

cerebellar decompression, but it appeared to be just as audible over the right as over the left side.

The impression gained from the operation was that undoubtedly a congenital arteriovenous aneurysm existed between the left vertebral artery and unknown veins.

Second Operation.—Two weeks after the first operation, the left vertebral artery was ligated between the atlas and occiput. The artery was perhaps three times its normal size. It was with great difficulty that it could be surrounded by a ligature and tied. Before the vertebral artery was ligated, the cerebellar

wound was bulging and tight; immediately afterward it became soft and somewhat sunken, and remained so during the remainder of the patient's stay of a month in the hospital. A discharge note by Dr. Deryl Hart states that the bruit, formerly heard so strongly over both temples, could be heard only over the right (contralateral) temple. The gait was much improved; the Romberg, Babinski and Oppenheim signs were negative. Subjective diplopia still persisted.

Two and a half years later, the patient wrote that he had remained well and free from headaches, but, judging by his letter, there had not been any mental improvement.

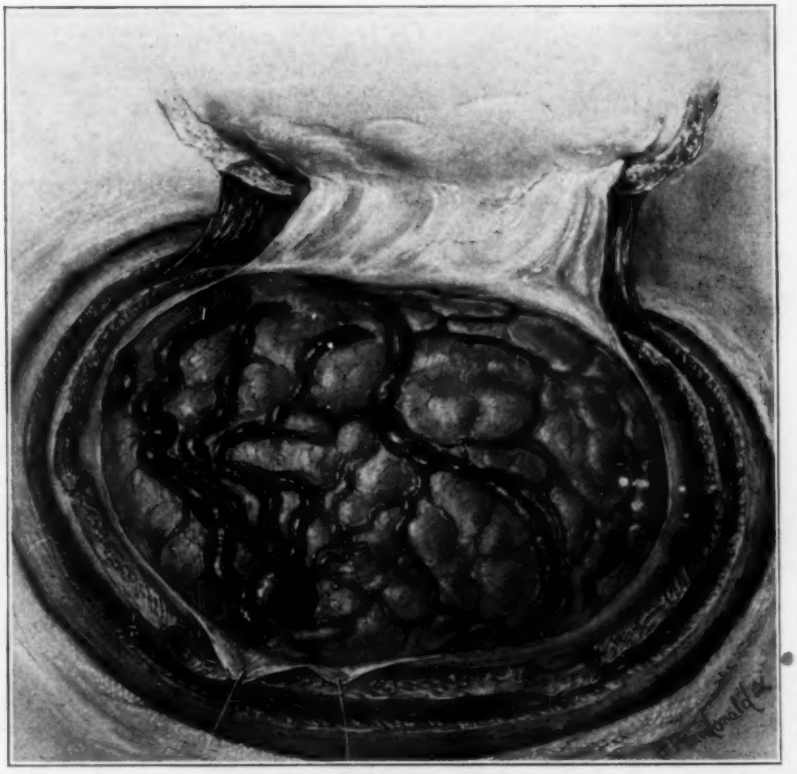

Fig. 10 (case 4).—A spider-like arrangement of veins abnormal in both size and position are directed toward the central swelling, which is just above and underlying beneath, and which can be palpated.

Case 4.—*History.*—A sparely nourished man, aged 47, was referred by Dr. L. E. Stubbs and Dr. H. W. Curtis, of Newport News, Va., on Oct. 1, 1925, because of frequent jacksonian convulsions and some motor weakness. Six years before this, he was suddenly seized with a cramp in the left hand; this was immediately followed by weakness of the legs, loss of consciousness and a convulsion. He regained consciousness an hour and ten minutes later, but was so weak that he could not leave the bed for three days. A month later, he was seized with an attack similar in origin but less severe; consciousness was lost for only a few minutes. During the next three years, such attacks occurred with fair regularity

about every two months. Three years before coming to Dr. Stubbs and Dr. Curtis, he had the first attack in which he was conscious throughout. A cramp attacked the left hand, causing his hand to close into a fist; the arm quickly became rigid and drew up above the head. He had a sensation of being unable to catch his breath; he thought it grew dark about him, and it seemed as if his heart had stopped beating; profuse perspiration broke out. He thought he was going to die. He thought his left leg was paralyzed, for he could not move it; the muscles of his trunk did not seem to be affected. This attack lasted half an hour, gradually passing away. For the next two years, there were no

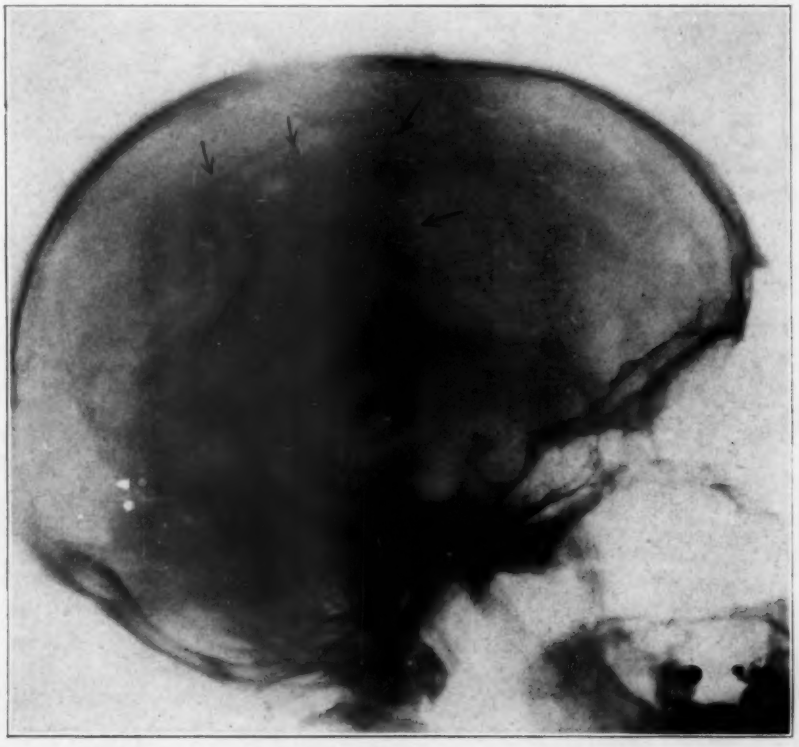

Fig. 11 (case 4).—Roentgen-ray observations. The arrows are directed toward the enormous shadows, and the whorl-like calcification of the vessel in the aneurysm is the most conspicuous shadow in the group.

more attacks (he was taking phenobarbital); then followed an attack like the last one. Since then similar attacks, without loss of consciousness, had occurred at intervals of about two months. After one attack the left arm and leg were paralyzed for half an hour, the function gradually returning. In addition, the left arm steadily became weaker, until it was considerably disabled. There had been only one attack at night, and no petit mal attacks. Severe headaches occurred after the attacks; with these exceptions, he did not have headaches. The attacks appeared to be induced by constipation, but not by exercise or excitement. Phenobarbital was definitely helpful; he ascribed the two years' free interval from attacks to this drug. There was no history of trauma.

Examination.—The neurologic examination showed that there was some motor weakness of the left side of the face and of the left arm, but not of the leg. Sensation was not changed. Biceps and triceps reflexes were markedly increased on the left side. The knee jerks were slightly greater on the left; the Babinski sign was negative. The Wassermann reaction of the blood was negative.

A roentgenogram of the head showed a group of calcified areas in the right frontoparietal region. In the anteroposterior view, these extended almost from the surface to the midline. The shadows were linear and circular, and in one place there was a definite whorl. The shape of these shadows suggested a vascular pattern, and from the roentgen-ray observations alone, a vascular tumor was suspected.

Fig. 12 (case 4).—Patient after the cerebral exploration and after the ligation of the internal carotid artery on the affected side. The partial occlusion later became complete by the reaction around the rubber band which was used to restrict it.

Operation.—On Oct. 3, 1925, a left craniotomy was performed over the region so clearly defined in the roentgenograms. The inner table of the bone was much thinner. These changes of pressure were due to a localized dilatation along the course of a large tortuous cerebral vein in front of and below the face portion of the rolandic area. From this venous center four large tortuous veins radiated as from the hub of a wheel (fig. 10). This venous dilatation was also situated almost in the center of an indurated subcortical mass, sharply defined by palpation and measuring about 5 by 5 cm. The large venous dilatation could be seen to pulsate, and when partially obliterated a thrill was imparted to the palpating finger. At each heart beat, the swirling red (arterial) blood could be seen through the walls of the vein. There seemed no hope of a successful

surgical intervention for such a vicious arteriovenous lesion. A subtemporal decompression was made; it remained fairly full and tight after the operation.

Second Operation.—Three weeks later, it was decided to ligate the right common carotid artery. Because of the danger of this procedure at the patient's age, a partial occlusion was first made with a rubber band. Twelve days later, the wound was reopened, the artery was ligated above and below the band, and the intervening tissue (including the band) was excised. There was a rather extensive reaction of all the tissues contiguous to the rubber foreign body. The carotid artery was already completely occluded with firm fibrous tissue within its lumen. It was our impression at the time that this was a satisfactory method of inducing a gradual occlusion of the carotid. It would, however, be dangerous if eventual total occlusion of the internal carotid artery could not be tolerated. No untoward symptoms were noted at the time of partial ligation or subsequently when it became total.

When the carotid artery was exposed for ligation, a lumbar puncture was performed, and the height of spinal fluid was measured. On each temporary closure of the carotid artery (to test for possible symptoms of cerebral anemia), the spinal pressure became reduced from 280 to 255 mm. and returned to the previous normal level when the lumen of the carotid artery was again restored. The blood pressure rose from 168 to 175 each time the carotid artery was closed.

For five days after the operation, the decompression was markedly sunken, then becoming flush with the skin.

The convulsions disappeared for more than a year. They then returned in mild form, but after eighteen months had again become like the former attacks.

CASE 5.—*History.*—A well nourished man, aged 48, was first seen in coma in the medical service of the Johns Hopkins Hospital, on Jan. 21, 1925. During the past ten years, he had had four convulsive seizures with loss of consciousness and with involvement of the left arm and leg. He did not rouse from the attack he had just before his admission to the hospital. He had incontinence of urine and feces, a pulse rate of 44 and rapid, stertorous breathing. Gradually, the coma disappeared. A residual partial hemianesthesia, hemiplegia and homonymous hemianopsia were present on the left side when consciousness was restored. Eventually, all of these signs disappeared almost entirely. Bloody fluid was obtained from several lumbar punctures. He convalesced slowly, and when discharged from the hospital six weeks after the onset of coma, he was mentally confused and forgetful. There was no history of trauma.

Six months later (July, 1925), he had another attack, with unconsciousness, but it lasted only three or four minutes. Shortly afterward, he returned to the hospital for treatment. During the time since his comatose spell he had done some work, but had been morose and gloomy. After dinner he sat alone or went to bed, always shunning company. He was forgetful and at times confused.

Examination.—A slight homonymous defect was found in the left visual field; the left knee jerk was obtained only on reinforcement; the right, not at all. No motor or sensory disturbance remained on the left side. The blood pressure was 125 systolic and 85 diastolic. The Wassermann reaction of the blood was negative.

It was evident that the patient had had a cerebral hemorrhage in the parieto-occipital region. At his age, and with negative vascular signs, it was safe to assume the existence of an underlying tumor or aneurysm from which the hemorrhage had arisen.

A ventriculography showed that in the right postrolandic region there was a great defect which was in free communication with the body of the lateral ventricle. The loss of cerebral tissue was interpreted to be the sequal of the hemorrhage which had occurred seven months before. The actual tumor or aneurysm did not show, but was assumed to be alongside the cerebral defect.

Operation.—On July 21, 1925, a right craniotomy was perfo ed. A plexus of large, tortuous veins obscured the brain just above the sylvian vein and below and posterior to the motor face area. The rolandic vein, which skirted the mass of superficial veins, was two or three times its normal size; the sylvian vein was also much enlarged. This circumscribed mass of veins dipped

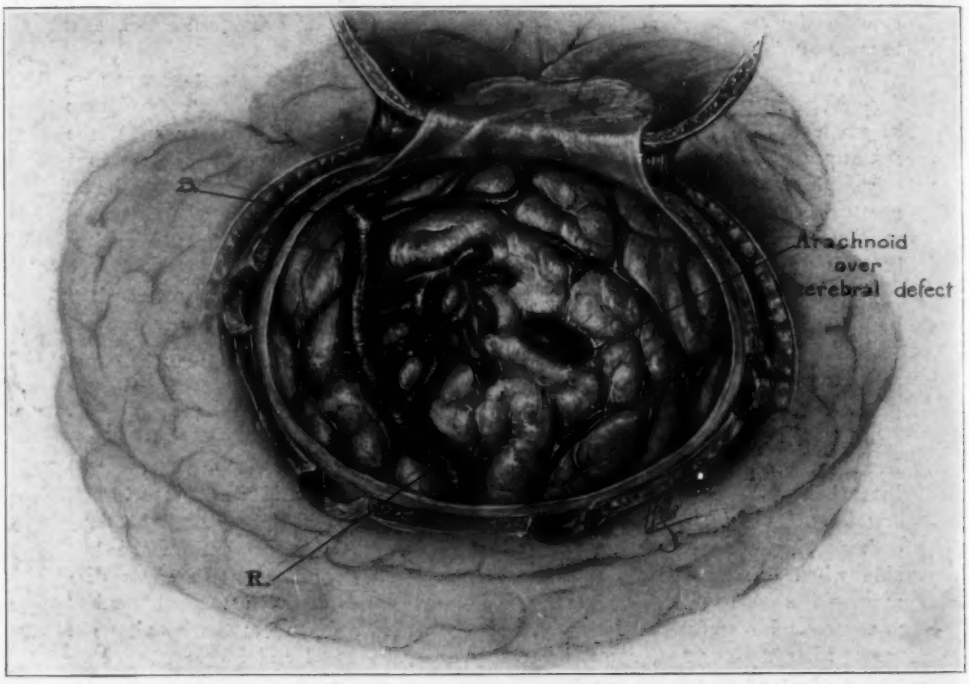

Fig. 13 (case 5).—The cluster of veins overlying and draining the mass of vessels below the surface of the vein. These veins abnormal in position empty into the tremendous rolandic vein which is continuous with an equally large sylvian vein. Arterial blood could clearly be seen in the veins. The cyst just posterior to the cluster of veins is the outer part of the defect which resulted from the cerebral hemorrhage which occurred several months previously.

into the cerebral cortex. Red arterial blood could be seen swirling in the rolandic vein with each heart beat. A thrill could not be felt.

About 1 cm. behind the venous plexus the meninges were transparent in an oval area about 1 by 1.5 cm. A cystlike dome was bulging upward from below. When this was opened, it proved to be the big ventricular dilatation (cerebral defect) indicated by the ventriculograms. The cerebral defect became larger below the surface and was nearly as large as a hen's egg. Its size was little reduced where it opened into the lateral ventricle.

Fig. 14 (case 5).—Reconstruction of arteriovenous aneurysm from the necropsy specimen. The large middle cerebral artery terminates in the mass of vessels — the so-called angioma — whereas on the opposite side the middle meningeal artery continues on posteriorly. The vascular bed protrudes into the defect which has resulted from the cerebral hemorrhage and which communicates freely with the lateral ventricle.

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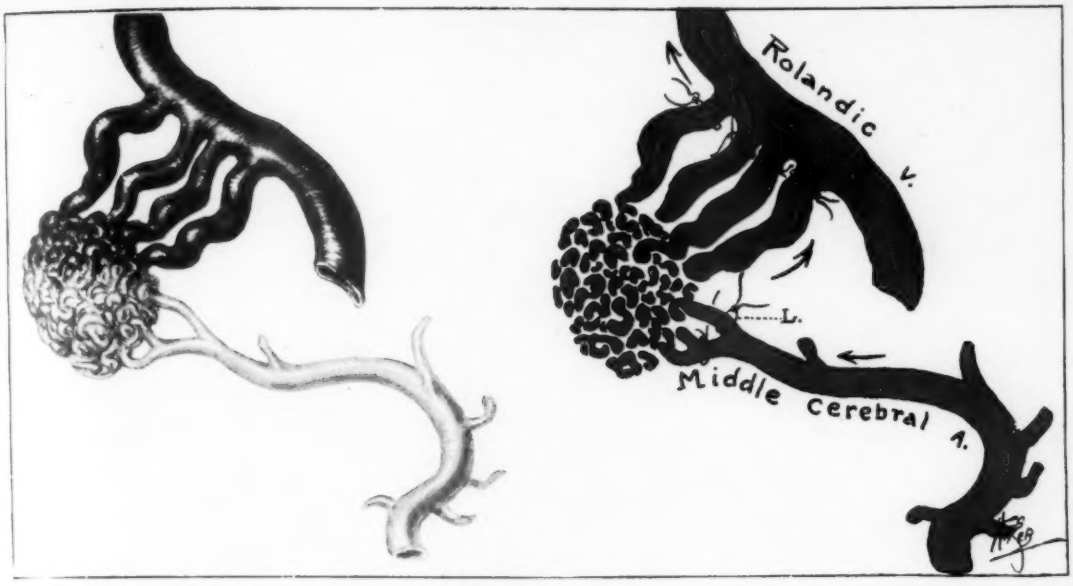

Fig. 15.—Diagram to show the construction and vascular arrangement of the arteriovenous aneurysm (c[illegible] V, fig. 14). The vascular intracranial mass takes the place of the former capillary bed from w[illegible]h it doubtless is a congenital maldevelopment.

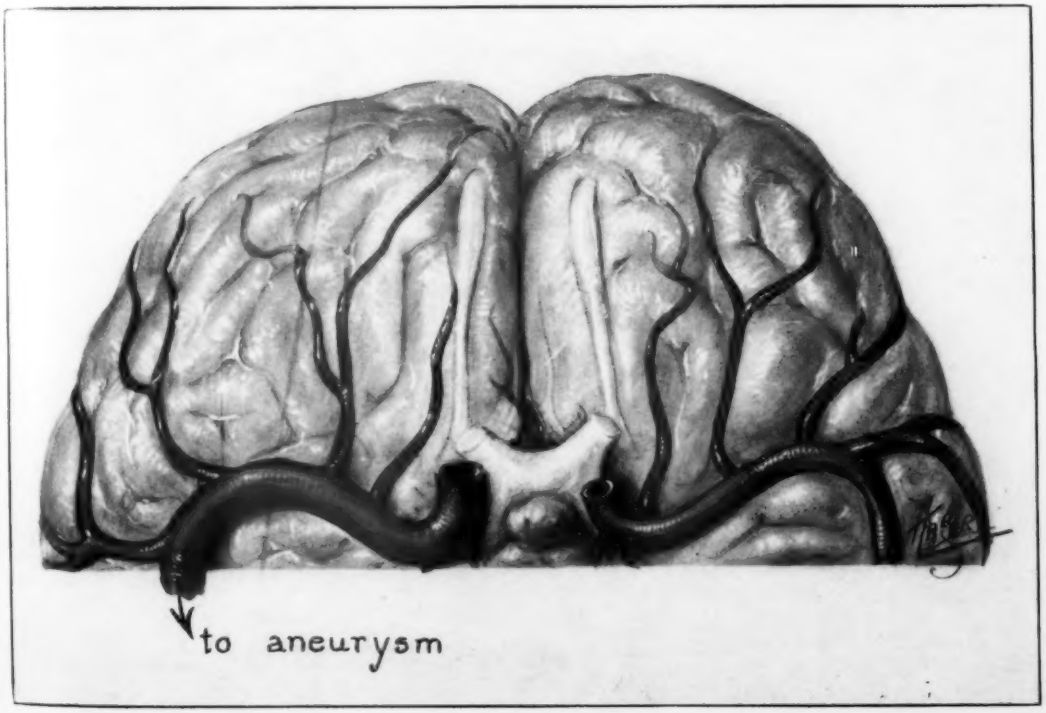

Fig. 16.—Sketch to show the relative size of the middle cerebral arteries on the normal and affected sides. The artery on the side of the aneurysm is considerably larger all the way to its origin in the carotid.

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In the anterior wall of the defect, several small, hard, dark gray nodules, from about 1 to 2 mm. in diameter, protruded into the cavity. Poorly covered by the white matter of the brain, they were recognized as vascular buds and as the outposts of a vascular tumor. Extirpation of the mass seemed possible and was attempted after ligation of the vessels on the surface of the brain. We were not unmindful of our earlier tragic experience in ligating the superficial veins, but this tumor looked far less vicious from the surface. After the tumor was circumvented by ligatures, extirpation was begun from the cyst forward. The cortex was first incised below the tumor in order to get the entering arterial trunks.

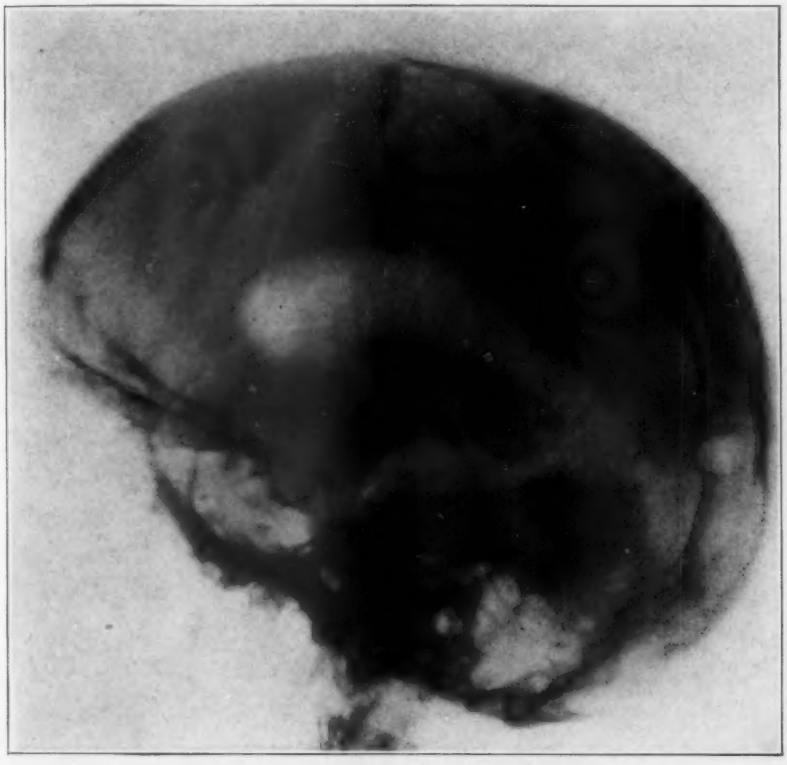

Fig. 17 (case 5).—Ventriculogram; a lateral view of the normal side.

A dense mass of intertwined serpentine red vessels of more uniform size was encountered.

A detour of the incision was made downward, and in this way the vascular mass was divided. Three fair-sized arteries were isolated near their entrance into the cluster of vessels. After ligation of the arterial supply, the vascular tumor was removed without difficulty. Bleeding was entirely stopped when closure was begun. However, despite a prompt recovery from the anesthetic, the patient died six days after the operation from a slowly developing intraventricular hemorrhage. During the operation the opening into the lateral ventricle had been carefully packed to preclude bleeding into it.

Necropsy showed three branches of the left middle cerebral artery clipped at the margin from which the tumor had been excised. Had it not been for the

open ventricular system, this patient would surely have survived, for the hemorrhage would have been far less significant and easily handled. Our reasons for excising this tumor were: 1. The tumor was back of the rolandic area, and a hemiparesis could probably be avoided. 2. Owing to the protrusion of the arterial units into the cerebral defect, a cerebral hemorrhage would probably occur again and by flooding the ventricular system cause death.

The middle cerebral arteries of both sides were dissected after death. The right was from one-third to one-half larger, even to its origin at the carotid

Fig. 18 (case 5).—Ventriculogram of the affected side. The large shadow is the cerebral defect which has resulted from the old hemorrhage. It will be noted that the volume of air which filled the left ventricle was only enough to fill the defect in the right ventricle.

artery. The affected middle cerebral artery turned upward and terminated in the branches which entered the nest of vessels. The left artery, however, took an entirely different course in this region and for some distance continued backward toward the occipital lobe.

CASE 6.—*History.*—A well nourished young man, aged 31, complained of loss of vision, progressive weakness of the left arm and leg, headache and convulsions.

The first symptoms of onset, dating back fifteen years, were intermittent spells of blurred vision and headache across the forehead on the right side. These

spells occurred once or twice a week. Limitation of vision to either side was not noticed at that time. In 1916, a decompression and puncture of the corpus callosum were performed at the Mayo Clinic. The patient was benefited for a short time, but the symptoms gradually returned.

In 1918, while on duty in France, he fell from a horse; a severe headache developed, but lasted only for several days. Craniotomy was performed at the Mayo Clinic in 1920. At that time, a choked disk was reported, and at the opera-

Fig. 19 (case 5).—Low power microscopic view of the network of vessels in the so-called angioma. These vessels vary tremendously in size and in the thickness of their walls, some contain elastic tissue of irregular strands; others do not. There is much thrombosis and irregular thickening of the intima.

tion, unusually large cortical vessels were noted. For the past six years, he has steadily become worse. His vision has steadily diminished; since 1920, he has been unable to see to the left side (left homonymous hemianopsia). The sense of pressure in the head, and particularly in the right temple, has become more and more intense. Numbness and weakness in the left hand, arm and leg began about 1921, and have gradually increased.

Epileptic attacks appeared soon after the sensory and motor symptoms. They were initiated with numbness in the left arm and leg and a drawing sensation in the left side of the face, but consciousness was not lost. The attacks usually came while he was asleep and awakened him. A painful headache always followed. In some of the attacks, only part of the left side was involved. The epileptic attacks steadily increased from two or three a month in 1920 to fifteen a month. The later attacks began with numbness and stiffness of the fingers of the left hand and the toes of the left foot. The right side was never involved. Diplopia appeared recently. During the past three years the vessels over his face and head, particularly on the right side, have become much larger and more conspicuous.

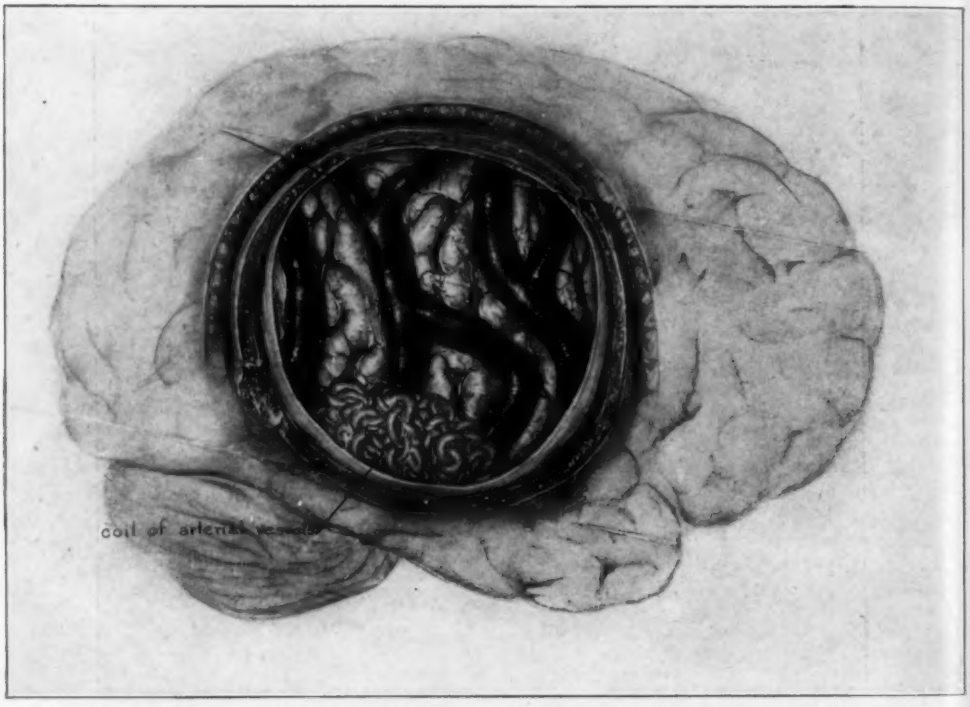

Fig. 20 (case 6).—Drawing of aneurysm as seen at operation. The interlacing vessels in the subdural space are clearly arterial. The large veins on the cortex of the brain drain the lesion which has doubtless extended into the brain.

Examination.—Over the occipital region of the right side was a diffuse bulging mass about 5 by 5 cm. but without sharply defined boundaries; it pulsated strongly with each heart beat. The superficial temporal, posterior auricular and occipital arteries were tortuous, much enlarged and easily visible. When all three of the arteries were compressed, the swelling in the scalp subsided and ceased to pulsate. A bony opening could be palpated just mesial to the right mastoid process and in the normal position of the mastoid vein. This opening, which also showed clearly in the roentgenogram, was about 1 by 1 cm. in diameter. Another small opening in the right frontal region was all that remained of a gradually closing decompression (performed ten years before). When the patient stooped, the veins

of the head and face became enormous. A loud systolic murmur was heard over the entire head, but much more over the right parietal and occipital regions.

The positive neurologic observations were: high grade papilledema (6 diopters) in both fundi; hypesthesia for pain, touch and temperature over the left side of the face and of the left arm and leg; slight weakness of the left facial muscles. The patient walked with a decidedly spastic hemiplegia gait; the left hand and arm were weaker than the right; sharply defined and complete left homonymous hemianopsia was present.

The corneal reflex was less active on the left; the knee jerks, biceps, triceps and achilles reflexes were exaggerated on the left; there was ankle clonus on the left; the Babinski sign was negative.

A roentgenogram showed a circular opening (1 by 1 cm.) in the occipital bone just mesial to the right mastoid process.

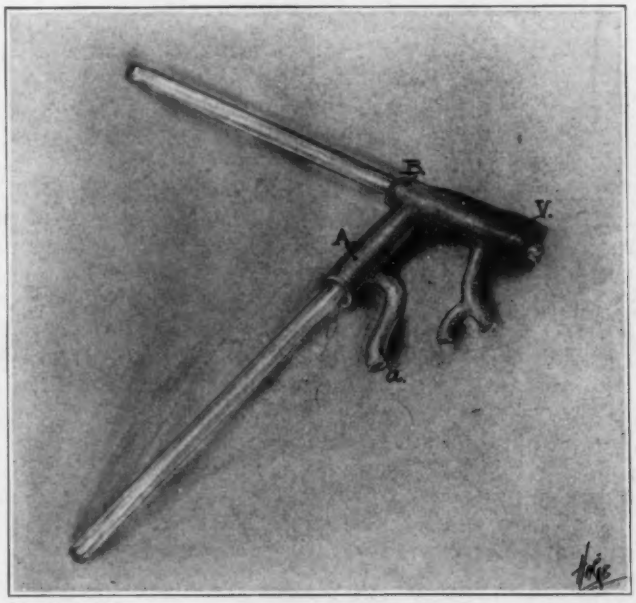

Fig. 21 (case 6).—Arteriovenous aneurysm of the scalp. The upper glass rod passes from the occipital artery into the mastoid vein (*V*). The posterior auricular artery (*A*) also is seen passing into the occipital artery near its junction with the mastoid vein. The mastoid vein entered a hole as large as one's finger to the left of the mastoid process. The superficial temporal artery (?) enters at the line of communication between the artery and veins.

Operation.—June 20, 1926: Since the extensive pulsating mass in the left occiput seemed to be dependent on the patency of the superficial temporal, the posterior auricular and the vertebral arteries, these were first ligated, although it was evident that the intracranial lesion (presumably also an arteriovenous aneurysm) was much more extensive, since there was hemianopsia, partial hemianesthesia and hemiplegia. The mass of vessels in the scalp was carefully dissected, and at the time the branches of the external carotid artery were exposed for ligation. All three of the vessels entered the extracranial vascular mass, and through the large, bony opening which corresponded in position to the mastoid

vein, a vein of corresponding size entered, filling the opening, and carried the blood from the vascular tumor. The angioma was dissected from the scalp and its entering arteries carefully marked for later study. The huge vein was carefully ligated at the bony opening with a transfixion suture of silk. Immediately after the operation, each vessel was carefully traced. The occipital artery passed directly into the big mastoid vein. This continuity was demonstrable by a probe which passed without interruption from the artery into the vein. Likewise a probe in the posterior auricular artery passed directly into the occipital artery just before it entered the big mastoid vein. A third small artery, presumably a branch of the superficial temporal, could not be traced accurately. There was, therefore, an arteriovenous fistula between the occipital artery and the mastoid vein and another interarterial fistula between the posterior auricular and the occipital arteries. The position of the great mastoid vein suggested that it entered the lateral venous sinus; it, therefore, seemed probable that the external arteriovenous fistula was separate and distinct from the arteriovenous aneurysm within the cranial chamber. Whether they were in communication through the lateral sinus could not be determined.

Second Operation.—July 20, 1926: The patient's signs and symptoms had been unchanged after the external operation. Feeling that an attack on the intracranial aneurysm would be too hazardous, a large right subtemporal decompression was performed in order to relieve the intracranial pressure. It was hoped that the patient might be relieved, at least to the extent of stopping the headaches and preserving the vision which was being menaced by a high papilledema.

When the dura, which was excessively bloody, was cautiously opened, several tortuous vessels lying in the subdural space were found attached to the inner side of the dura. Most of these vessels could be stripped away from the dura, but others entered it and had to be ligated. These extremely tortuous vessels were red and pulsated strongly. All that were visible were clearly arterial channels.

These arteries, which formed free loops in the subdural space, could be compared only to a tangled mass of angle worms; they were also about that size and fairly uniform. This vascular tumor was situated along the posterior part of the temporal lobe. Everywhere this part of the brain was completely hidden from view. Three tremendous veins emerged from this cluster of vessels. A fourth vein of about the same size paralleled the anterior part of the decompression and could not be seen to enter the angioma. Pulsation could be seen and a thrill palpated in these veins.

When the vessels were freed from the dura, furious bleeding resulted. It was possible to control the hemorrhage only by a cotton pack. The next day, careful removal of the pack was again followed by arterial bleeding, though somewhat less severe. A piece of muscle then controlled it.

The headaches were entirely relieved by the operation, but the symptoms and signs have remained as before. The decompression has since bulged at times, but at other times has been sunken.

Case 7.—*History.*—A normal looking boy, aged 14, was referred by Dr. Ralph Greene, of Jacksonville, Fla., because of jacksonian epilepsy (Oct. 27, 1926). His trouble dated back to the age of 7 years, when his mother handed him a glass of water; he dropped it and said his hand was weak and that he could not hold it. He was conscious throughout the attack. A few weeks later when cutting paper, he complained that he could not use his right hand. Three months later, when at school, he had the first convulsion. After the attack, he recalled that his tongue

felt queer and that he could not swallow. It was observed that his right hand was drawn in the convulsion. A second convulsion occurred a year later; it resembled the first. A month later, the right side suddenly became completely paralyzed. Though he was unable to talk, he understood everything. There was some fever for three or four days and severe headache. A week later, the function began to return in the leg and arm, and in three weeks speech began to return. In three or four months, his speech was again normal, but there has since remained a slight residual weakness of the arm and leg. Further symptoms did not appear until two weeks before he came to the Johns Hopkins Hospital

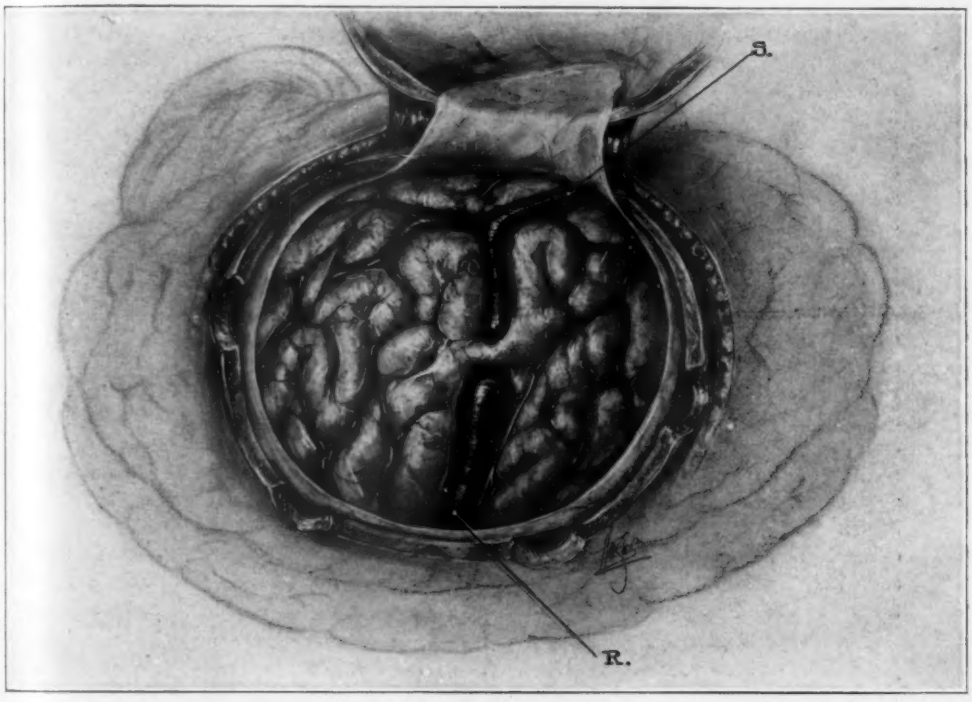

Fig. 22 (case 7).—The large rolandic vein can be seen coming directly out of the brain and has arisen from the vascular mass situated beneath. Arterial blood could be seen pumping into it. It will be noted that it has no connection with the group of veins below and which also comes out of the depth of the brain. The convolutions separate these two ends of the vein. The lower branch is continuous with the sylvian vein which is also large.

(October, 1926), when another attack began in the right hand. He had headaches only after attacks. There was no history of trauma.

Examination.—The neurologic examination showed the following positive symptoms: definite weakness of the facial muscles, the right hand, arm and leg; some atrophy of the right side; increased deep reflexes on the right; no ankle clonus, and a positive Babinski sign on the right.

The roentgenogram was negative. The Wassermann reaction of the blood was negative.

A diagnosis of tumor or aneurysm was made, more probably the latter because of the old history of a cerebral hemorrhage and with so little progression of signs or symptoms over a period of seven years.

A filling defect nearly obliterated the anterior part of the body of the left lateral ventricle. In the background of air, a linear calcified shadow was clearly visible, whereas in the roentgenogram without air, this shadow could not be seen.

Operation.—On Oct. 29, 1926, left craniotomy was performed. When the dura was exposed, a localized bluish swelling was evident toward the mesial part of the bony defect. The dura over this region had been unusually bloody; it was also thinner than elsewhere. The localized bulge was due to an underlying venous dilatation which appeared to come out of the brain tissue and which passed into a large vein that coursed mesially to the superior longitudinal sinus. It corresponded in position with the rolandic vein, which it undoubtedly represented. A second smaller vein passed downward from the mass to the sylvian vein. The large rolandic vein was distinctly red, and through its thin walls the pulsing red blood could be seen rhythmically mixing with the darker venous blood. No other enlarged veins were present on the surface of the brain. Palpation of the brain revealed an indurated area beneath the face area, but no attempt was made to expose the underlying tumor or to treat the lesion in any way. Its position in the important part of the leading cerebral hemisphere precluded any further effort.

A year later, his condition was practically unchanged.

CASE 8.—*History.*—A well nourished man, aged 35, in good general health, complained of generalized convulsions (May 5, 1925). His first attack had occurred fourteen months earlier. It was described as a generalized convulsion which came without warning and lasted about half an hour. He frothed at the mouth, bit his tongue but did not have incontinence of urine. The attack came on during sleep. A few hours later, he had another attack which seemed to be similar to the first, and two hours later another. Three months later, he had another attack at dinner, and was told that his left side had been rigid (probably incorrect). This attack lasted from twenty to thirty minutes. Additional attacks occurred six, ten and fifteen months later. Unilateral features were not noted in any of them except the one just quoted, but he has been told that his head usually turned to the right. There was no history of trauma.

Neurologic and Physical Examinations.—The results of the neurologic and physical examinations were negative. The Wassermann reaction of the blood was negative.

The ventricular system was dislocated to the right side, indicating a cerebral tumor on the left. The ventricular fluid was normal.

Operation.—On July 21, 1925, left craniotomy was performed. Five greatly dilated and somewhat tortuous veins stood out in strong relief on the surface of the brain. The rolandic and sylvian veins formed a relatively enormous continuous channel from the sylvian fossa to the superior longitudinal sinus. An almost equally large offshoot of the sylvian vein paralleled the rolandic vein and entered the longitudinal sinus, and two other branches of similar size passed downward from the sylvian vein to enter the transverse sinus. The latter veins, running closely together, formed en route a plexus over the temporal lobe. The red blood could be seen swirling in these veins with each pulse beat and mixing with the darker venous blood. The arteriovenous connection was deeper in the brain and could not be seen or palpated. The wound was closed without any attempt to treat the aneurysm.

Second Operation.—Two weeks later, the left common carotid artery was exposed under local anesthesia. After several transient compressions of the carotid

without subjective or objective noticeable change, the artery was ligated just below the junction of the internal and external carotid arteries. Manometeric readings of the spinal fluid were made at the time of compressions of the internal carotid artery (lumbar puncture was performed at this time). With each temporary closure of the common carotid artery, the pressure of the spinal fluid fell from 240 to 215 mm. The pressure returned to its former level when the arterial compression was released. The blood pressure rose from 104 to 110 immediately after the ligation.

It should be noted that for several days before the ligation, the carotid artery was compressed on many occasions by pressure on the neck and without symptoms; the results indicated that the operative ligation would probably be safe.

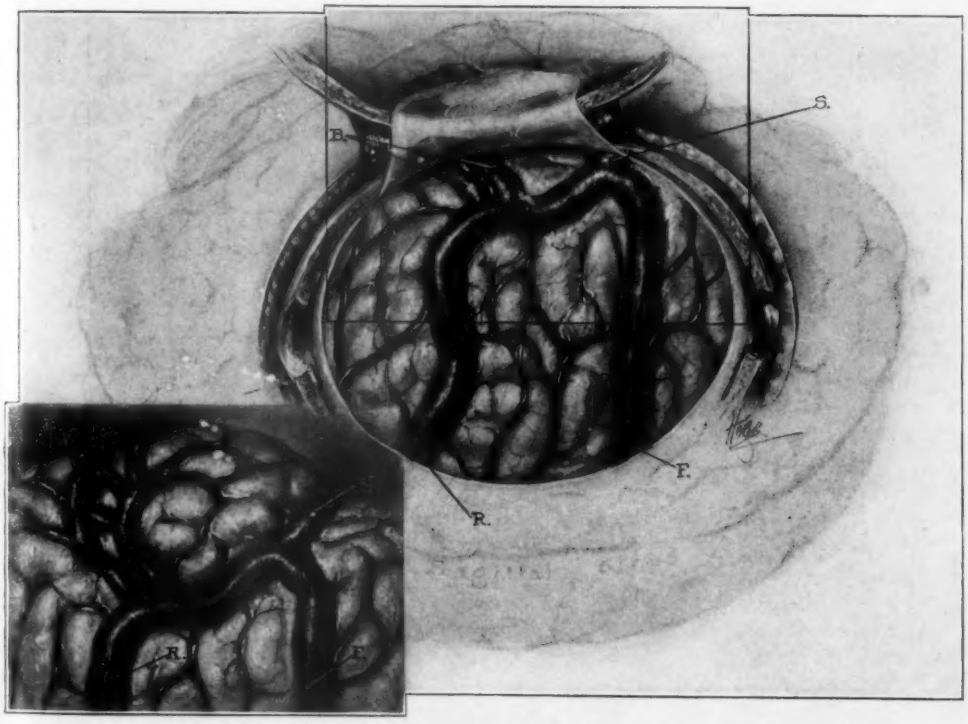

Fig. 23 (case 8).—Showing the distribution of the large distended veins on the surface of the brain and also the cluster of veins over the temporal lobe. Arterial blood could be seen pumping into these tremendous channels. As this aneurysm involved the most important part of the brain no attempt was made to treat the aneurysm directly. The left internal carotid artery was subsequently ligated. *R* indicates rolandic vein; *S*, sylvian vein; *F*, frontal vein; *B*, veins to lateral sinus.

The patient has remained free from both petit and grand mal attacks to date (two and a half years). There have been no untoward symptoms as a result of the closing of the common carotid artery.

Extracranial Arteriovenous Aneurysm—the Prototype of the Intracranial Lesion.—A well nourished, healthy woman, aged 29, was referred by Dr. A. E.

Pagan, of Washington, D. C., because of deforming veins of the scalp. Since early childhood (at least since she was 2 years of age), a lump had been present in the midline of the scalp and between the parietal eminences. It had always pulsated, but had been soft and painless. It had not given her any trouble, and had not grown in later years. Nine years before, when her first child was born, a lump, thought to be a big vein, appeared in the middle of the forehead. This swelling grew forward and became larger spasmodically; during each of seven pregnancies, it increased in size until it overhung the bridge of the nose and extended over to the inner canthus of each eye. Many other large tortuous veins

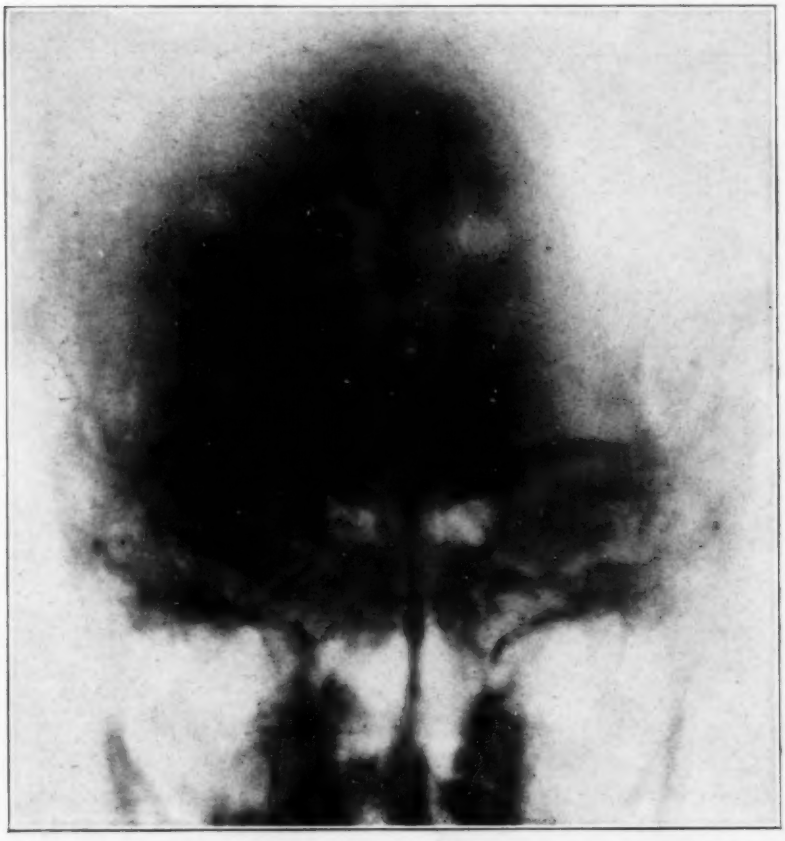

Fig. 24 (case 8).—Ventriculogram showing the small left anterior horn as compared with the normal on the right.

filled the scalp and became steadily larger until they began to encroach on the outer canthus of both eyes. The patient again became pregnant and was anxious about the vein increasing in size at the time of delivery. There had been no signs of intracranial pressure, though intermittent general headaches had been present for four or five years. On one occasion she had had an attack of numbness involving the right hand and lasting two weeks, but consciousness was not lost. There was no motor weakness during the attack, or at any other time. Noises in both ears had appeared recently and were somewhat annoying.

Examination.—A pulsating tumor about as large as a lemon was palpable in the midline of the head between the parietal eminences. When the whole head was shaved, this mass was easily visible. From this tumor, as from the hub of a wheel, a number of enormous, tortuous veins coursed posteriorly, laterally and anteriorly (fig. 27). Pressure on the jugular veins promptly caused them to become tight and much fuller. Pressure on certain veins caused the distal portion to collapse, but the collapsed portion nearly always filled up slowly from collateral channels; when the collateral channels also were compressed, the distal part of

Fig. 25 (case 8).—Patient after ligation of the internal carotid artery in the neck.

the veins remained collapsed and did not fill (fig. 28). Particular reference is made to this point because of the large midline vein which extended to the nasion. When the collapsed venous space was palpated, it felt rough and one received the impression that there was a groove in the bone; until this test was made we wondered whether there might not be communication with the superior longitudinal sinus situated immediately beneath. The possibility of this communication was,

therefore, eliminated by the test. Palpation of the veins failed to disclose an arterial pulsation. A slight murmur could be heard over the veins when examination was made with a stethoscope. A thrill could be definitely made out over the large median vein and in other veins near the tumor. The greater the distance from the tumor, the less distinct the thrill became. The degree of pressure must

Fig. 26.—Arteriovenous aneurysm of the scalp which doubtless had intercranial extensions since the patient had localized sensory involvement of the right hand at one time. This lesion is a prototype of the intercranial aneurysm shown in figure 20. A localized mass posteriorly corresponds with the so-called angioma.

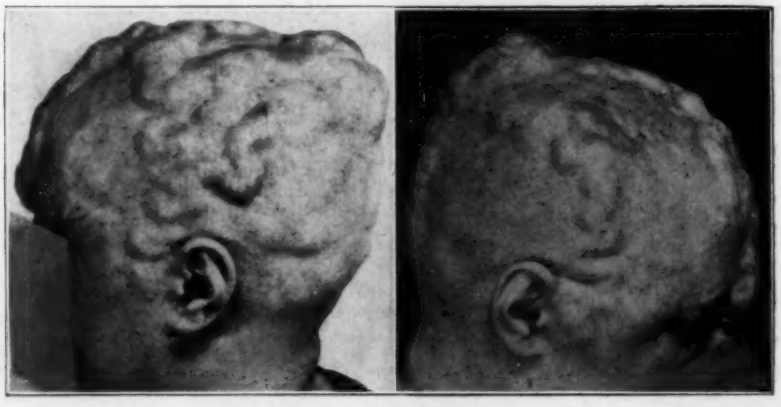

Fig. 27.—Same as patient in figure 26.

be just right to elicit the thrill. All of the veins were easily compressed. The tumor mass itself gave a strong expansible pulsation. The tumor was fairly soft and partly compressible, giving the impression of a mass of angle worms. When the mass was compressed firmly, it became painful and induced headaches. Compression could not be tolerated for more than a few minutes, nor could a number

of veins be compressed at one time without causing pain and headache—doubtless for the same reason. Temporary occlusion of the veins caused the central tumor to become full and tight.

The roentgenogram showed large grooves of the middle meningeal artery on both sides; both grooves extended to the midline and appeared to end directly beneath the tumor of the scalp, otherwise the roentgenogram was negative.

The results of the neurologic examination were negative. The Wassermann reaction of the blood was negative.

A diagnosis of arteriovenous aneurysm of the scalp was made. The large channels of both middle meningeal arteries and the focal numbness of the hand made it seem probable that there was an intracranial extension of the aneurysm.

Operation.—The tumor itself was not removed, for intracranial extension was feared, because of the numbness of the hand which had been present for two

Fig. 28.—The collapse of the vein over the forehead when the arterial supply is cut off from the vascular mass. The venous channels then become greatly sunken.

weeks. The superficial temporal, internal maxillary and occipital arteries were ligated on both sides of the neck. On the left side, the external carotid artery was also ligated at its origin. After ligation of the arteries, the veins responsible for the tumor on the forehead were ligated. The veins responsible for this part of the swelling had previously been determined by compression with the fingers. The disfiguring part of the venous bed at once disappeared and has not returned. The redundant part of the skin disappeared and no sign of the old deformity remained. A few days after the operation, all of the veins in the scalp became hard and tender and were no longer compressible. Apparently they had thrombosed, and when the patient was seen six months later, all trace of them had disappeared. The central tumor mass had greatly diminished in size and no pulsation could be detected. The tinnitus had ceased. The patient had been well in every respect.

TABLE 1.—*Analysis of Author's Eight*

Case, Age, Sex*	Location of Vascular Tumor	Duration of Symptoms	First Symptom	Principal Symptoms	General Pressure Signs and Symptoms	Focal Symptoms	Roentgen-Ray Observations	Eyegrounds
I 52 ♂	Right temporal lobe	6 yr.	Convulsion (general)	Convulsions (grand and petit mal); headache (1 week); soreness right temple (1 week)	Headache (1 week)	Head turns to left in convulvulsion; some subjective numbness in fingers of left hand	Negative	Normal
II 19 ♂	Right parietal lobe	4 yr.	Convulsion	Jacksonian convulsions often followed by weakness of left hand	None	Jacksonian convulsions beginning in left side of face	Negative	Normal
III 35 ♂	Cerebellar left vertebral artery	2 yr.	Staggering gait	Staggering gait, diplopia, headache, dizziness, dysarthria; mental impairment	Headache; choked disk; hydrocephalus	Staggering gait, Romberg, deafness	Negative	Choked disk
IV 47 ♂	Fronto-parietal right	6 yr.	Focal convulsions	Convulsions; motor loss on left side	None	Left hand, arm and leg involved in convulsion; motor weakness left arm	Extensive shadows in right fronto-parietal region	Normal
V 48 ♂	Parieto-occipital right	10 yr.	Convulsion	Convulsions; cerebral hemorrhage, moody and forgetful	None, except during cerebral hemorrhage from which he recovered	Left side involved in attacks; left paresis, anesthesia, and hemianopsia after cerebral hemorrhage, later disappeared	Negative	Normal
VI 31 ♂	Temporo-occipital	15 yr.	Transient visual disturbances	Headache, convulsions, hemiplega, hemianesthesia, hemianopsia, diplopia	Choked disk; headache	Hemiplegia, hemianesthesia, hemianopsia, focal convulsions (left side)	Large circular hole in right occiput	Choked disk
VII 14 ♂	Parietal	7 yr.	Attack of weakness in right hand	Convulsions (jacksonian); weakness right side of body; symptoms for cerebral hemorrhage on one occasion	None	Weakness right side of face, right arm and leg; attacks beginning in right arm; motor aphasia and hemiplegia after cerebral hemorrhage	Linear calcified shadow	Normal
VIII 35 ♂	Left parietal	14 mo.	Convulsion	Convulsions	None	Head turns to right	Negative	Normal

* In this table and the following tables, ♂ indicates male; ♀, female.

Headache	General Epilepsy	Focal Epilepsy	How Diagnosed	Diagnosis before Operation or Autopsy	Operations and Observations	Conditions at Necropsy	Remarks
1 week only	+	Head turns to left	Ventriculography and craniotomy	Tumor of the brain	Anomalous branch of right middle cerebral artery supplied vascular mass; after its ligation, mass of venous vessels collapsed	Living	Patient has had but one attack since operation (3½ years); never had headache; general health excellent
None	..	+ begins in left face and arm	Craniotomy	Tumor of the brain	Mass of serpentine vessels with large emerging veins; attempted extirpation; vessel burst between the ligatures	.....	Patient died of hemorrhage
+	None	None	Neurologic signs of localization; cerebellar exploration	Tumor of the brain	(1) Cerebellar exploration; (2) ligation left vertebral artery	Living	Patient's general pressure and neurologic signs and symptoms largely disappeared, but mental changes persisted
None	..	+ left arm	Roentgenogram	Aneurysm of the brain	(1) Exploration of aneurysm; (2) internal carotid later ligated	Living	Attacks returned after a year's absence following operation; the shadow in the roentgenogram was the important diagnostic sign in this case
None	+	+	Ventriculography	Tumor or aneurysm (because of cerebral hemorrhage)	Excision of vascular tumor	Shows arterial trunk from middle cerebral supplied tumor	Cerebral hemorrhage from the vascular mass
Severe	..	+	By external arteriovenous vascular bed	Arteriovenous aneurysm	(1) Extracranial arteriovenous aneurysm (dissected out); (2) mass of angle worms in temporo-occipital region; big veins (in cortex) with arterial pulsation	Living	Two separate arteriovenous aneurysms present, one in the scalp, the other in the brain
None	..	+	Ventriculography, history and exploration	Tumor or aneurysm	Big venous dilatation in which arterial blood could be seen	Living	Patient gave history of a cerebral hemorrhage, one year after first symptoms
None	+	Head turns to right	Ventriculography	Tumor of the brain	(1) Large distended rolandic and sylvian veins; arterial blood seen (through wall of vein) to be pumping into venous blood; venous plexus over temporal lobe; (2) internal carotid artery ligated	Living	Has had no attacks to date (2½ years)

Fig. 29.—Greatly enlarged meningeal grooves, both right and left, directed toward the vascular bed of this aneurysm (interior set of arrows). The posterior set of arrows show the greatly enlarged venous channels in the skull providing exit for the blood leaving the vascular tumor, i. e., to the lateral sinuses.

Fig. 30.—Result two weeks after the operation at which the occipital, posterior auricular, internal maxillary and superficial temporal arteries were ligated on both sides, and in which the venous channels were ligated at the two points indicated by the pressure of the fingers in figure 28. Several months after this photograph was taken all the veins had subsided and the pulsation in the arterial mass had ceased.

In retrospect, there is reason to believe that removal of the mass might have been safely performed without ligation of the branches of the external carotid. The result, however, would not have been different.

SEX AND AGE AND TIME OF APPEARANCE AND DURATION OF SYMPTOMS IN CASES OF ARTERIOVENOUS ANEURYSM OF THE BRAIN

In the literature, twelve of the cases of arteriovenous aneurysm of the brain reported occurred in men and eight in women. Curiously, the eight patients in the cases reported in this paper were men, but there seems to be no reason to lay emphasis on this point or to expect any differences due to sex.

The time of appearance of convulsions or the other more occasional symptoms is one of the most surprising features of this lesion. Being surely of congenital origin, it would be logical to expect symptoms soon after birth. Data on this point are available for eighteen of the thirty cases as follows: The first symptoms appeared in seven patients between the ages of 1 and 10; in three patients between 11 and 20; in three patients between 31 and 40; in five patients between 41 and 50; there was no record of first symptoms for patients between 21 and 30 years of age.

In 44 per cent of the patients, therefore, the first symptoms did not appear until after the age of 30 and in 30 per cent, symptoms began after the fortieth year. The latest time of appearance was at the age of 48.

The duration of symptoms ranged from a few hours to fifty years. In four of eighteen cases, symptoms had been present for more than twenty years.

LOCATION OF INTRACRANIAL ARTERIOVENOUS ANEURYSMS

The aneurysms are located in almost every part of the brain. Three are in the cerebellar region (Leunenschloss,[27] Znojemsky,[28] Dandy). In one case (Znojemsky) there were two separate and distinct aneurysms: one in the right cerebellar lobe, the other in the cerebral hemisphere. In two instances, there were arteriovenous aneurysms in the scalp, apparently independent of the intracranial lesion (Mühsam, pulsating exophthalmos; Dandy, occipital artery and mastoid vein).

An overwhelming proportion of the aneurysms are located in the paracentral region and have arterial connection with a branch of the middle cerebral artery. In two cases, the corpus callosal artery supplied the arterial connection (Deetz,[29] Steinheil [10]).

27. Leunenschloss, O.: Ueber das Angioma arteriale racemosum des Gehirns, Studien zur Pathologie der Entwicklung **2**:21, 1914.

28. Znojemsky, J.: Angioma arteriale racemosum arteriae cerebelli inferior, anterior, sinistrae et Angioma racemosum arteriae cerebri mediae sinistrae, Rev. v neurol. psychiat., fig. a diaetet. therap., v Praze **7**:207, 1910.

29. Deetz, E.: Ueber ein Angioma arteriale racemosum in Bereich der Arteriae corpus callosi, Virchows Arch. f. path. Anat. **168**:341, 1902.

THE GROSS APPEARANCE OF ARTERIOVENOUS ANEURYSMS

The venous trunks draining the aneurysms (when located in the cerebral hemisphere) nearly always connect with the external venous circulation through the rolandic and sylvian veins, and when these form a continuous channel, as they usually but not always do, both show the maximum effect of the arteriovenous connection. This is evidenced by their dilatation throughout their course. The degree of involvement of the contiguous veins varies in individual cases; at times other nearby veins may be almost unaffected. In a single case, the venous outlet was through the internal venous system (Steinheil); one small vein of Galen and the great vein of Galen were dilated and tortuous throughout their course to the sinus rectus. In addition, a sacculated aneurysm with a long pedicle was present on the vein of Galen.

Because the external venous system carries the outflow of blood from most of the arteriovenous connections, the characteristic venous changes are on the surface of the brain. This makes it possible to detect these cases so easily at operation. And since the veins are free from all accessory coverings, it is also possible to see through their walls and detect the pulsing red, arterial blood against a background of black venous blood. This opportunity does not exist elsewhere in the body. So far as I am aware, attention has not been directed to this point—so essential to the diagnosis—heretofore.

In many of the cases of arteriovenous aneurysms located in the cerebral hemispheres, there are congenital abnormalities of the venous system on the exterior part of the hemisphere. In one case, the rolandic vein arose directly out of the brain substance, having its origin in the aneurysm. In two other cases, the rolandic vein did not exist as a well defined venous channel.

The degree of venous engorgement is dependent on the size of the arterial supply and on the size of the mass of vessels, or rather on the size of their lumina, forming the arteriovenous communication. A large angioma with many vessels takes some of the brunt of the arterial force off the venous channels of exit. A small angioma or a short connecting link throws all the arterial force directly into the veins. The smaller the artery and the smaller the lumen of the arteriovenous communication (i.e., the larger the number of vessels intervening between the artery and veins), the smaller will be the venous channels which drain the aneurysm. And conversely, the larger the artery and the larger the lumen of the arteriovenous connection (i.e., the smaller the mass of vessels between the artery and vein), the greater will be the effect on the veins emerging from the aneurysm. Many large veins, therefore, indicate a small angioma or a free end to end communication between an artery and vein. Smaller veins indicate a larger angioma and an arteriovenous

communication which is relatively well compensated, i. e., nearer the function of the capillary system. Doubtless there are many smaller arteriovenous aneurysms with a small arterial inlet and relatively small veins of exit which could not be established as abnormal. A thrill could not be felt in these, nor could arterial blood be seen pumping into them. There have been several cases at the Johns Hopkins Hospital which showed definite vascular abnormalities and which were thought to be arteriovenous aneurysms, but these have been excluded from all consideration because the absolute evidence was lacking.

Kaiserling's [30] case and case 1 in my series are excellent examples of excessively large and tortuous veins which result when no barrier is interposed between an artery and a vein. In Kaiserling's case, a tortuous elongated artery could be seen emptying directly into a huge venous chamber. In case 1, a short anomalous branch of the middle cerebral artery, and with normally thick arterial coats, abruptly passed into a vein. The transition between the thick arterial and the thin venous coats was clearly seen during the inspection of the vessel. Reid's case [31] of an arteriovenous aneurysm between the subclavian artery and veins in the neck seems in every way an exact duplicate of this case in the brain.

A mass of vessels forming the connection between the artery and veins, i. e., the so-called angioma, when such is present, is usually in the depths of the brain, but this is not necessarily true. In case 6, the mass was external, at least in large part. The gross character of the mass of entwined vessels is also variable; some look exactly like arteries, some like veins, and some like a combination of arteries and veins.

The arteries supplying the mass of vessels have always been larger than normal; some have been very tortuous (Deetz,[29] Simmonds,[16] Kaiserling [30]). In the only case of my series in which the artery was observed, the middle cerebral artery was enlarged all the way to the internal carotid artery. It was not tortuous, nor was its enlargement excessive—perhaps from one-third to one-half larger than the corresponding vessel of the opposite side (fig. 16). The arterial change, while always definite, is never comparable to that of the veins, which is really the characteristic part of the picture of arteriovenous aneurysms.

HISTOLOGIC APPEARANCE OF THE VESSELS IN SO-CALLED ANGIOMA

The histologic appearance of the vessels comprising the vascular skein is varied. The vessels show extreme differences in size, even in the same

30. Kaiserling: Ueber ein ungewöhnliches Aneurysma kortikaler Hirnarterien, Verhandl. d. Patholog. Ges. **16**:220, 1913.

31. Reid, M. R.: Studies on Abnormal Arteriovenous Communications, Acquired and Congenital, Arch. Surg. **10**:601 (March) 1925, fig. 12; **10**:996 (May) 1925; **11**:25 (July) 1925; **11**:237 (Aug.) 1925.

section. The walls are at times thin, suggesting veins, and others alongside are thicker, suggesting arteries. The intima may be narrow, but frequently it is greatly thickened and at times irregularly so. Many vessels are entirely occluded by thrombus formation. The elastic fibers are shown in special stains to be present in the intima of many vessels but not in others. Rarely is the elastic tissue a well defined layer; usually a strand is present here and there. The media is usually poorly developed, with much hyaline degeneration or calcification. These changes, while doubtless varying considerably in detail, are essentially similar in all microscopic reports in the literature. They indicate abnormal, poorly developed vessels with debilitated walls, which render the person the victim of vascular rupture long before the allotted time for normal vessels. The extensive thrombosis has been commented on by several authors (Emanuel,[32] Therman,[33] Leunenschloss,[27] Astwazturoff,[9] Kaiserling [30]). It is doubtless one of the important reasons for the progression of the lesion in the later years of life. Each thrombosed vessel throws an additional strain on the venous system and on the angiomatous mass. Doubtless it is this added strain which is also responsible for many of the hemorrhages. The intercommunication of the vessels within the vascular nest has been described. I have been unable to learn whether or not this was true in case 2 which came to necropsy, for the mass was mutilated during its removal. Simmonds [16] reported evidence of canalization of thrombi.

SIGNS AND SYMPTOMS OF ARTERIOVENOUS ANEURYSM OF THE BRAIN

(*a*) *Convulsions.*—With four exceptions, the first symptom in this group of cases was a convulsion, and most of these (all but two) were of the jacksonian type. The starting point of the seizures, i.e., the aura, when present, varied with the exact point at which the motor or sensory tracts were involved. It is, of course, not necessary that the attacks show the jacksonian march; at times even the focal or unilateral character of the attacks may be none too clear, though probably in all cases there will at least be turning of the head to the opposite side. Jacksonian attacks are present only when there is a direct infringement on the motor tracts. Those aneurysms at a distance from the motor tracts will give less conspicuous unilateral signs in the attack, and usually no aura. In two of the cases reported here, there were no warning signs (aura). In both of the cases without jacksonian convulsions the head

32. Emanuel, C.: Ein Fall von Angioma arteriale racemosum des Gehirns, Deutsche Ztschr. f. Nervenh. **14**:288, 1898.

33. Therman, E.: Ein Fall von Angioma racemosum cerebri, Arch. a. d. path. Inst. d. Univ. Helsingfors **3**:67, 1910.

turned away from the side of the aneurysm and eventually in each other unilateral features presented.

In a number of instances, consciousness was not lost in the attacks even when the seizure involved an entire side of the body. (Deist,[34] Laves,[35] Steinheil,[10] Dandy). Later, as the attacks became more severe, the other side of the body also became involved and consciousness was lost.

Indeed convulsions need not occur even though the motor or sensory areas are involved. Only two cases in which the cerebral hemispheres were involved failed to cause epilepsy (Simmonds,[16] Struppler[15]). Two cases of cerebellar arteriovenous aneurysms involving the cerebellum failed to produce convulsions (Leunenschloss,[27] Dandy), but tumors in the cerebellum or the posterior cranial fossa rarely cause epilepsy,

(*b*) *Motor and Sensory Disturbances.*—Another important and frequent sequel of the convulsions is a transient sensory or motor paralysis. This occurred in nearly all of the cases which have been reported. Sensory or motor weakness following a convulsion is strong evidence in favor of a tumor of some kind. When the same motor involvement occurs repeatedly with such precision of attack and over a period of so many months or years and with little or no permanent progression, an arteriovenous aneurysm would appear to be the most probable lesion. The paralysis often does increase slowly in these cases, for the volume of the vascular network must slowly increase, especially in later life. It will be remembered that the degenerative and proliferative changes (calcification and thrombosis) which occur prematurely in the angiomatous mass must from time to time demand readjustments and always the volume of the network of vessels must expand in this process, thereby causing increased paralysis.

Sudden hemiplegia or hemianesthesia are the results of hemorrhage, which is so prone to occur at any age, but particularly during advancing years. Usually the paralysis disappears, in part or whole, the amount of recovery depending on the proximity of the aneurysm to the motor or sensory tracts.

(*c*) *Other Focal Signs and Symptoms.*—Other focal manifestations are at times present. Transient attacks of aphasia occurred in the cases of Ranzel[36] and Sterzing.[37] Homonymous hemianopsia was noted by

34. Deist, H.: Ein Fall von Angioma racemosum der linken Lobus paracentralis in seiner klinischen und versicherungsrechtlichen Bedeutung, Ztschr. f. d. ges. Neurol. u. Psychiat. **79**:412, 1922.

35. Laves, W.: Ein Fall von Angioma arteriale racemosum des Gehirns, Jahrb. f. Psychiat. u. Neurol. **44**:55, 1925.

36. Ranzel, F.: Zur Kasuistik kombinierter Hirnaffektionen; ein Fall von Rankenangiom des Gehirns mit tuberculous Meningitis, Wien. klin. Wchnschr. **22**:1214, 1909.

37. Sterzing, P.: Ein Fall von Angioma arterial racemosum im Gehirn, Centralbl. f. allg. Pathol. u. path. Anat. **19**:278, 1908.

Steinheil [10] and was present in two of the cases in my series, one permanently, the other only transiently after a cerebral hemorrhage. Simmonds [16] reported blindness in one eye and loss of smell and taste. The reflexes on the contralateral side of the body are, of course, always increased when a motor weakness exists; often ankle clonus and usually a positive Babinski are also present under the same conditions. Deist's patient had a positive Babinski sign, even though motor weakness was not apparent.

Mental changes have been uncommon. Steinheil's patient became demented. One of the patients in my series became moody and was forgetful after recovery from a cerebral hemorrhage.

(*d*) *Cardiac Changes.*—Hypertrophy of the heart has been shown to occur in dogs after the production of arteriovenous fistulas (Reid,[5] Holman [7]). Some of the cases reported by Reid and Holman also showed cardiac hypertrophy and even decompensation, for which they assumed the arteriovenous fistula to be responsible. Since enlargement and tortuosity of the arteries always follow a fistula, it is easy to believe that the heart too must suffer. Holman produced measurements to indicate that the size of the heart was actually reduced after the cure of an arteriovenous fistula. Emanuel,[32] Isenschmid [26] and Laves [35] found cardiac enlargement in their cases and attributed it to the effects of the aneurysm. The heart was not found to be enlarged, or its function disturbed in any of the cases reported here.

(*e*) *Signs and Symptoms of General Pressure.*—In only seven of the thirty cases was there a history of headaches (Mühsam,[12] Simmonds,[16] Eiselsberg,[13] Emanuel,[32] Dandy, [three]). This is noteworthy when the great size of the lesion is considered. The space adjustment, however, has occurred in early life and only gradual changes usually occur thereafter, excluding, of course, the always overhanging possibility of a sudden hemorrhage. In one of my patients, a man of 52, a headache had been present for only a week prior to the patient's admission to the hospital; his headache was sharply localized to the right frontal region owing to the fact that the vascular bed was in contact with the meninges. The headaches in other cases have been generalized. The patient of Campbell and Ballance and three of the patients in my series had severe headaches after each attack. Emanuel's patient was excused from military duty because of severe headaches which persisted throughout youth and finally disappeared; his head was also large.

Few examinations of the eyegrounds are reported in the assembled cases. A papilledema is reported only by Mühsam. The eyegrounds were carefully examined in all of our cases. In six, there was no change; in two, a well advanced choked disk. The pressure in one of these was due to an obstructive hydrocephalus; the cerebellar lesion had

blocked the aqueduct of Sylvius. In the other case, a choked disk had been present for more than ten years. After a subtemporal decompression, the decompression was at times full and tight, again soft and even sunken. The great variations in pressure may account for the preservation of vision even with such a long-standing swelling of the eyegrounds.

Diplopia as a sign of pressure was reported only twice (Mühsam, Dandy).

(*f*) *Cerebral Hemorrhage.*—The premature degenerative vascular changes which have been commented on explain the frequency of cerebral hemorrhage in this group of cases. It is a fairly safe, but not absolute, rule that when a cerebral hemorrhage occurs during youth or middle age, i. e., before the time when vascular accidents are to be expected, there must be an underlying tumor or aneurysm, either of which will contribute defective vessels. In the twenty-two cases collected from the literature, nine patients, or 41 per cent, died of cerebral hemorrhage (Drysdale,[38] Kaiserling,[30] Borchardt,[39] Ranzel,[36] Simmonds,[16] Sternberg,[40] Sterzing,[37] Wichern [41] and Leunenschloss [27]). Only two of the eight patients whose cases are reported herewith had a cerebral hemorrhage, one known and the other presumed from the history, but both patients recovered. The probability of death from cerebral hemorrhage from these lesions is also increased because in many instances the nest of vessels frequently projects into the lateral ventricle. A hemorrhage of smaller size may develop in the tumor or the contiguous brain tissue and may not induce coma, as in one of the eight cases; the hemorrhage seemed certain because of the sudden complete hemiplegia with slow recovery. Sterzing's case is a remarkable example of the capricious character of the defective vessels in the vascular network. His patient, who died of cerebral hemorrhage at the age of 32, had suddenly developed hemiplegia with disturbance of speech (presumably due to a cerebral hemorrhage) twenty-two years earlier when 10 years old. After a slow return of function, there had been no motor change until a year and a half before death, when the same side again became paralyzed; curiously, jacksonian convulsions did not develop until after this late attack of paralysis. Childbirth was an inciting cause of the cerebral hemorrhage in Borchardt's case.

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38. Drysdale, J. H.: Report of Case, Pathological Society of London, Lancet, 1904, p. 96.

39. Borchardt, M.: Die chirurgische Bedeutung der Gehirnaneurysmen, Beitr. z. klin. Chir. **133**:429, 1925.

40. Sternberg, C.: Demonstration eines Falles von Angioma arteriale racemosum des Gihirns, Verhandl. d. deutsch. path. Gesellsch. **7-9**:308, 1905.

41. Wichern, H.: Klinische Beiträge zur Kenntnis der Hirnaneurysmen, Deutsche. Ztschr. f. Nervenh. **44**:220, 1912.

Roentgenologic Evidence.—The results of the roentgen-ray examination were of great importance in two cases. In one case, a probable diagnosis of an intracranial aneurysm was made principally on the basis of the roentgen-ray examination. A series of calcified shadows covered an area roughly circular and about 5 cm. in diameter; all were beneath the surface of the brain. While most of the shadows had no unusual shape, circular or spiral, the shape of three of this group suggested calcium deposition in the walls of the blood vessels. In fact, such shadows could hardly be produced in any other lesion (excepting of course calcified blood vessels in tumors).

In a second case, a linear shadow, 1.5 cm. long, could be seen along the anterior horn of the lateral ventricle. This shadow, however, was visible only after the ventricle had been filled with air, which afforded an excellent contrast medium for the background. In a third case, a circular hole about 1 cm. in diameter, located alongside the mastoid, was clearly shown in the roentgen ray. The importance of this roentgenographic evidence, however, was minimized because it was readily palpable, being large enough to admit the tip of the index finger.

The large middle meningeal grooves (bilateral) commented on in the history of the extracranial arteriovenous aneurysm cannot be used as positive evidence because a cranial exploration was not made. As almost all of the intracranial aneurysms are located within the brain, there is little possibility of any vascular burden being referred to the middle meningeal arteries, and therefore the arterial vascular changes which are usually sought from this source in roentgenograms of the skull will be lacking. The positive roentgen-ray observations in cases of arteriovenous aneurysms of the brain will be largely confined to the calcified deposits in the abnormal vessels of the brain, or perhaps to localized areas of erosion. It is noteworthy that in two of the cases reported here, there was a localized erosion of the inner table of the skull, but the roentgenograms failed to disclose it.

VENTRICULOGRAPHY

The use of the air contrast medium in the cerebral ventricles is always important when the existence or even the character of an intracranial lesion is in doubt. Even though arteriovenous aneurysms only occasionally produce signs and symptoms of intracranial pressure, the lesion is one which occupies intracranial space, and therefore will usually give the positive evidence of a deformed ventricular system. Mention need not again be made of the calcified deposit which could be observed only with an air background, but such a condition is of less importance than the deformation of the ventricular system.

In two of the cases in my series in which the unilateral character of the attack was rather uncertain, the ventriculogram showed the entire

ventricular system to be dislocated to the opposite side. The roentgenographic changes do not, of course, make the diagnosis of an arteriovenous aneurysm; they only denote a space occupying lesion. In another case, the ventriculogram indicated a large defect communicating with a lateral ventricle. Since the patient had previously had a history of cerebral hemorrhage, the ventricular hernia was assumed to be due to the localized hemorrhage which occurred on that occasion. But this cranial defect did not indicate the location of the aneurysm or tumor which had caused the hemorrhage.

Ventriculography yields much invaluable information in cases of epilepsy. It at once separates those lesions which are space-occupying. It will also demonstrate those lesions which are due to loss of brain tissue; also many of the congenital cerebral deformations. Even in cases of well defined focal or jacksonian epilepsy, ventriculography often yields most important information prior to a cranial exploration, for many lesions are below the cortex and would escape detection by inspection or palpation of the brain were the ventriculographic evidence not known at the time.

THE DIAGNOSIS OF ARTERIOVENOUS ANEURYSM OF THE BRAIN

The signs and symptoms of arteriovenous aneurysms show striking uniformity when taken as a whole. The outstanding complaints are: (1) jacksonian convulsions usually followed by (2) a transient motor or sensory disturbance, and (3) a gradually progressive and permanent motor or sensory paralysis coming on over a period of years and not directly depending on convulsions. When to these are added the long time element, the data are sufficiently characteristic at least to suggest the diagnosis of an arteriovenous aneurysm above other lesions affecting the brain. The transient paralysis following a convulsion and particularly the gradual permanent paralysis (not related to convulsions) makes a tumor formation of some kind an almost certain diagnosis. The accurately repeated jacksonian convulsions, involving such a sharply restricted part of the rolandic area and particularly when covering a period of so many years, should make a true neoplasm very unlikely. When the convulsions begin in early life and extend into the adult life, a neoplasm can almost be excluded. But when the epilepsy begins in later life and the time interval is still short, slowly growing tumors such as the meningiomas are important lesions to be differentiated; likewise they may show pressure symptoms late. Venous angiomas must always enter into consideration. They too will cause transient motor and sensory losses after the jacksonian seizures, but the permanent motor and sensory changes are far less possible, and when they do occur they are less progressive than in the arteriovenous variety. With symptoms, such as

those described in the typical cases, the diagnosis of arteriovenous aneurysm should at least always be strongly suspected.

Moreover, given sharply defined jacksonian convulsions, appearing in youth and continuing into adult life, with a gradually progressive

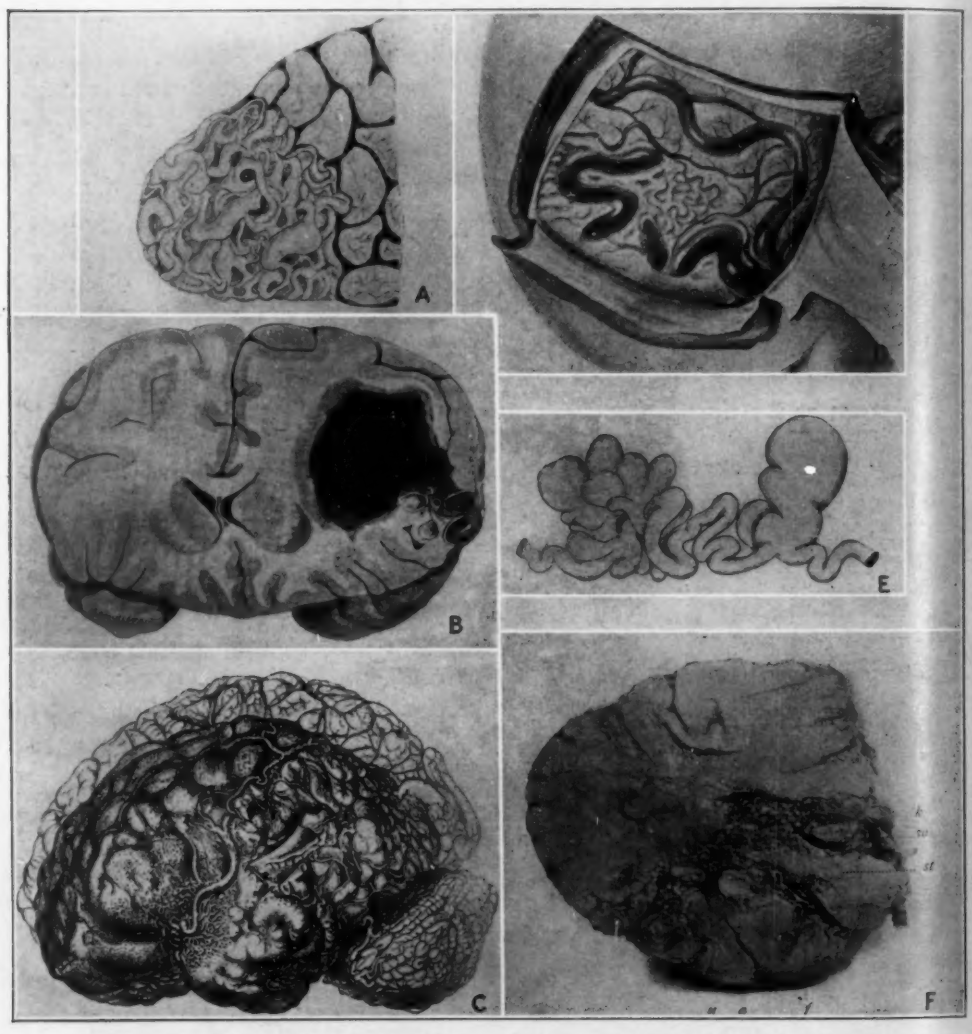

Fig. 31.—Group of specimens taken from cases reported in the literature. They show the variations which arteriovenous aneurysms may take in the brain. *A* indicates Simmond's case; *B*, Borchardt's case; *C*, Kalischer's case; *D*, Krause's case; *E*, Simmond's second case (the mass was dissected); *F*, Emanuel's case.

paralysis and then a superimposed cerebral hemorrhage such as afflicted so many of these patients, one could almost venture an absolute diagnosis of an arteriovenous aneurysm.

In those cases with the less well defined jacksonian convulsions developing in adult life and without a slow paralysis, one would have little reason to suspect an arteriovenous aneurysm; rather the diagnosis of a cerebral tumor would be considered most probable.

The roentgen-ray evidence, as in one of the cases which I have reported, may, unaided be sufficient for diagnosis. The presence of cal-

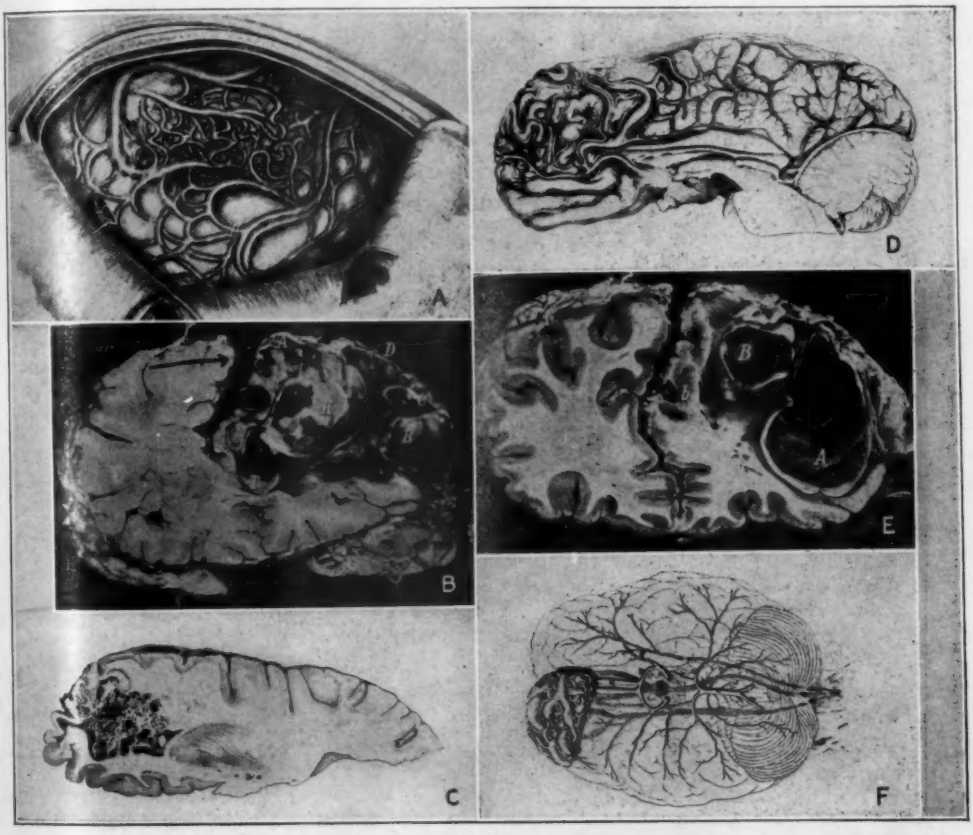

Fig. 32.—Group of specimens from the literature. *A* indicates Campbell and Ballance's case; *B*, Kaiserling's case. The arrow at the top of the brain is directed to the arterial venous connection which Kaiserling has marked *A;* the extensive thrombosis formation is marked *K*. *C* indicates Deetz's case showing the intracranial extension of the aneurysm; *D*, Deetz's case, in which the aneurysm arose from the corpus callosal artery; *E*, Kaiserling's case. This section is anterior to the one in *B*. *F* is taken from Steinheil's case in which the outlet of the aneurysm was into the vein of Galen.

cified shadows in circles or whorls, is, I believe, almost pathognomonic of vascular degeneration. When taken in conjunction with the clinical data, the diagnosis should be unequivocal.

Not infrequently a well defined extracranial arteriovenous aneurysm is demonstrable in patients who also have the intracranial signs and symptoms described for cerebral arteriovenous aneurysms. With such symptoms, an intracranial lesion of the same type is to be expected. An intracranial arteriovenous aneurysm was diagnosed in one of the cases reported here by this method of reasoning (case 6).

THE TREATMENT FOR ARTERIOVENOUS ANEURYSMS OF THE BRAIN

Occasionally spontaneous recovery in cases of arteriovenous aneurysm has been reported, but there is little real hope for such a successful outcome either in the brain or elsewhere in the body. The only way to cure an arteriovenous aneurysm is to ligate the entering artery or to excise the vascular tumor. But the radical attempt at cure is attended by such supreme difficulties and is so exceedingly dangerous as to be contraindicated except in certain selected cases. It has never seemed fair to make this attempt except on the insistent demand of the patient who fully understood all phases of the situation. Moreover, there are ways of helping the condition with little if any risk, and this in many cases at least seemed more to be recommended than the attempted extirpation of the vascular mass.

As in most cerebral lesions, however, each case should be considered a law unto itself. There are large aneurysms and small ones; those which are mostly arterial, others mainly venous; some are superficial, others deep; some are in highly important areas of the brain, others in portions largely silent. All of these factors, and finally the patient's wishes in the matter, must be weighed. An aneurysm in the left cerebral hemisphere in a right-handed person is surely noli me tangere under all conditions. Any attempted cure, even if successful, would almost surely result in disturbances of speech or motor power, or of both. Aneurysms of the corpus callosal arteries do not offer any greater surgical possibilities.

There is more reason to attempt to cure a patient who has arteriovenous aneurysms in the right cerebral hemisphere. In most of these cases, the arterial connections with the lesions are provided by branches of the middle cerebral artery. Should the aneurysm be postrolandic, the arterial connection can be ligated without injury to the motor function. In one case, we were able to do this without great difficulty. After ligating the arteries supplying the angioma, excision of the vascular bed was easy and relatively bloodless. In another case, in which the anterior pole of the right temporal lobe was covered with great tortuous and distended veins, the anomalous vessel was located and ligated with a silk ligature. It arose from the middle meningeal artery just beyond its origin from the internal carotid; following this the mass of distended

venous trunks collapsed. This patient has remained well for three and one-half years; he has had only one convulsion (a year ago), whereas before the fistula was closed, convulsions occurred every month. There is no assurance of a permanent cure of epilepsy even after the cure of an aneurysm. The cerebral defect will always remain at the site of the old lesion and any lesion of the cerebral hemisphere will always remain a potential source of convulsions. However, the convulsions will be far less and more easily controlled with an inactive lesion than with one which is active.

A third and only other attempt at radical cure by extirpation of the angioma was our first experience with a lesion of this type. Thinking at the time that it was a venous angioma, its extirpation after careful ligature of all the superficial veins seemed possible. The huge rolandic vein was doubly ligated and divided both above and below the great mass of cortical vessels and another vein was being sutured when suddenly the rolandic vein burst, not at the site of the sutures, but midway between them. It was then evident that the bleeding was arterial, and that with the venous outlets closed, the arterial pressure within the veins was greater than the wall of the vein would stand. The vein ruptured at the localized swelling shown in the drawing, i. e., at the weakest point and probably where the main force of the artery is projected. This accident demonstrated the danger of closing the veins while the arteries supplying the aneurysm are intact. When the second ligature was being placed on the big rolandic vein it was observed that considerable force was necessary to tighten the knot and occlude the vessel, and it could be done only step by step; the arterial pressure was responsible for this difficulty. Krause's case is interesting in this connection. He, too, ligated some veins on the surface, thinking the angioma to be venous and hoping to benefit the patient by reducing the number of veins. Fortunately nothing happened. He commented on the fact that the ligated veins "ballooned out like sausages." Bergmann's [42] patient died from uncontrollable bleeding; assuming a localized bulge in the dura to be a cyst, a blind puncture into it was followed by terrific bleeding. Laves' patient also was lost on account of hemorrhage following an exploratory puncture of the brain. Once there is a tear in such a nest of vessels, their defective walls almost preclude all chance of controlling the bleeding. Each attempt at hemostasis is promptly followed by more bleeding.

In two of my cases in which the aneurysm was located in the rolandic and prerolandic areas, palliative treatment was tried, the internal carotid artery being ligated in the neck. In one case there have been no attacks,

42. Bergmann: Zur Kasuistik operativer Hirntumoren, Arch. f. klin. Chir., **65**:935.

TABLE 2.—*Analysis of the Cases of Arteriovenous*

Author	Date, Sex, Age	Location	Duration Symptoms	First Symptom	Principal Symptoms	Sign or Symptom of General Pressure	Focal Symptoms	Eye-grounds
Bergmann[42].....	....	Fossa of Sylvius and paracentral fossa	..	.....	.....	.....	.....	.....
Borchardt[39].....	1925 ♀ 27	Paracentral	..	.....	Convulsions; paralysis	.....	.....	.....
Campbell and Ballance[14]	1922	Paracentral	..	Weakness of arm and leg; and fits	Mild degree hemiplegia and anesthesia; convulsions	None	Hemiplegia and hemianesthesia; increased reflexes; ankle clonus	.....
Deetz[29].....	1902 ♀ 56	Right side of corpus callosum	..	Convulsion	Convulsions; partial hemiplegia	None	Left hemiplegia; jacksonian convulsions	.....
Deist[34].....	1922 ♂ 56	Paracentral (left)	12 yr.	Convulsion	Convulsions	None	Jacksonian convulsions; Babinski +	Negative
Drysdale[38].....	1904 ♀ 26	Prerolandic (right)	11 yr.	Convulsion	Convulsions; weakness	.....	.....	.....
Eiselsberg[13].....	1913 ♂ 39	Paracentral	..	Convulsion	Convulsions; headaches	Headaches	Jacksonian convulsions	.....
Emanuel[32].....	1898 ♂ 36	Paracentral	30 yr.	Headaches when a boy	Convulsions	Headache; large head	Jacksonian convulsions	.....
Kaiserling[30].....	1913 ♂ 26	Most of hemisphere	..	.....	.....	.....	.....	.....
Kalischer[11].....	1897 ♂ 7	Sylvian fossa and frontal lobe	1 yr.	Convulsion	Convulsions; hemiplegia gradually became complete	None	Convulsions and hemiplegia	Negative
Krause[8].....	1908 ♂ 46	Paracentral	..	.....	.....	.....	.....	.....
Laves[35].....	1925 ♀ 53	Paracentral and postrolandic	12 yr.	.....	.....	.....	.....	.....
Mühsam[12].....	1924 ♀ 23	Occipital region	..	.....	Diplopia; headache	Diplopia; headache	.....	Large tortuous vessels; slight choking
Ranzel[36].....	1909 ♂ 36	Left occipital lobe	2 yr.	Convulsion	Convulsions; some motor and speech disturbance	None	Jacksonian convulsions, hemiplegia, aphasia	.....

Headache	General Epilepsy	Focal Epilepsy	How Diagnosed	Diagnosis Before Operation or Autopsy	Operation and Observations	Conditions at Necropsy	Remarks
..	..	..	Operation and necropsy	Brain tumor	Large veins on surface of brain	Walnut sized knot of vessels beneath surface of brain, very tortuous	Puncture of dura blindly; terrific hemorrhage, uncontrollable, death; diagnosed cavernous angioma
..	+ right	..	.....	.....	.....	Section shows aneurysm	Old specimen from Virchow Krankenhaus; patient died in childbirth of cerebral hemorrhage
After attacks	..	+ left toes	Operation	Tumor	Large veins on surface	Living	Hemiplegia and hemianesthesia were gradually progressive
..	..	+ left-sided	Necropsy	.....	.....	Right corpus callosal artery large and tortuous; mass of vessels plunged into frontal lobe	Very extensive tumor covers mesial surface of hemisphere, fills frontal lobe and reaches anterior horn of ventricle and corpus striatum
None	..	+ right leg	Necropsy	.....	.....	Large veins on surface	
..	..	+ arm and leg	Necropsy	.....	.....	Anterior cerebral artery very large; hemorrhage	Large veins on surface obscured brain; patient gradually became more and more deeply comatose following fit 10 days previously
+	..	+ right side	Operation	.....	Large veins on surface	.....	Very large veins on surface of brain; ligated; condition of patient unchanged
In youth only	..	+ arm	Necropsy	.....	.....	Large surface veins; mass of vessels in brain grown into ventricle and choroid plexus, also extended to pulvinar	Patient died in coma following attack; was excused from military duty because of headache; vessels are thrombosed and calcified; no mention of cerebral hemorrhage
..	..	+	Necropsy	.....	.....	Large surface veins	No history, except that patient died in coma following attack; this is a remarkable specimen, the veins and arteries being sharply defined; the communication is shown in the photograph; the venous channels are larger than in any other case; there is no network of vessels
None	..	+ right corner of mouth	Necropsy	.....	.....	Large veins on surface and mass of vessels in frontal lobe	No change in big arteries at base of brain; Virchow said it was an unusual case
..	..	+	Operation	.....	Ligated some veins, says they then distended like sausages	Large surface veins; smaller vessels of the tumor just reach the surface	Typical surface appearance of lesion shown in painting
..	..	+ left hand	Operation and autopsy	.....	Large veins on surface; no thrill or pulsation	Cluster of entwined vessels below surface	No loss of consciousness in earlier attacks; at operation brain was punctured, severe bleeding which was uncontrollable; branch of middle cerebral artery (containing small sacculated aneurysm) enters vascular tumor which protrudes into ventricle; hypertrophy of heart
+	..	..	Necropsy	.....	.....	.....	A pulsating tumor near inion connects with veins of brain and longitudinal sinus; also pulsating exophthalmos; very large veins in dura and in the brain and on its surface
..	..	+ right arm	Necropsy	.....	.....	Small knot of vessels in left occipital lobe	Patient died of hemorrhage in an attack

TABLE 2.—*Analysis of the Cases of Arteriovenous*

Author	Date, Sex, Age	Location	Duration Symptoms	First Symptom	Principal Symptoms	Sign or Symptom of General Pressure	Focal Symptoms	Eye-grounds
Simmonds[16].....	1905 ♀ 13	Right occipital lobe	6 yr.	Headaches	Headaches, hemiplegia, loss of touch and smell; blindness right eye	Headache	Hemiplegia	.....
Astwazaturoff [9].	1911 ♀ 35	Right fronto-parietal	..	Convulsion	Convulsions; headache	Headache	Jacksonian convulsions	.....
Simmonds[9].....	1905 ♂ 55	Right parietal	Since childhood	Convulsion	Convulsions	.....	.....	.....
Steinheil[10].....	1895 ♂ 49	Right frontal	Since childhood	Convulsion	Convulsions; progressive paralysis and hemianopsia (left homonymous)	.....	Hemiplegia; hemianopsia (left homonymous); jacksonian convulsions	Normal
Sternberg[40].....	1905 ♀ 25	Temporal lobe	..	.....	.....	.....	.....	.....
Sterzing[37].....	1908 ♂ 32	Corpus striatum	22 yr.	Weakness right leg	Hemiplegia; convulsions; some motor aphasia	No	Hemiplegia; jacksonian convulsions	.....
Struppler[15].....	1900 ♀ 48	Paracentral	Few hours	Coma	None until coma	No	None	.....
Wichern[41].....	1912 ♂ 32	.....	..	Hemiplegia	.....	No	Hemiplegia; jacksonian convulsions	.....
Leunenschloss[27]	1914 ♂ 24	Right cerebellar lobe	15 yr.	Headache	Headache; hemiplegia; deafness; dizziness	Headache	Deafness	.....
Therman[23].....	1910	Parietal lobe	..	.....	.....	.....	.....	.....
Znojemsky[28]....	1910	Parietal lobe also cerebellum	..	.....	.....	....	.....	.....

Aneurysms Collected from the Literature—Continued

Headache	General Epilepsy	Focal Epilepsy	How Diagnosed	Diagnosis Before Operation or Autopsy	Operation and Observations	Conditions at Necropsy	Remarks
+	None	None	Necropsy	.....	.....	Enormous dilated veins on surface; tumor of entwined blood channels	There was much thrombosis with some evidence of canalization; patient died of cerebral hemorrhage from attack
+	..	+ left side	Necropsy	.....	.....	Mass of vessels size of dove's egg below cortex right fronto-parietal region; enlarged veins on surface of brain	Patient died of meningitis; calcareous deposits in vessels of vascular mass
..	..	..	Necropsy	.....	.....	Specimen shows a large coiled vessel with many pouching dilatations; no note about veins	It is open to question whether this case is an arteriovenous aneurysm; patient died of cerebral hemorrhage (into ventricle)
..	..	+ left face	.....	Epilepsy; later, aneurysm	.....	Left corpus callosol artery large and tortuous; right vena galeni parva and vena galeni magna large; tumor consists of mass of entwined vessels	Patient finally became demented; vessels of tumor did not seem either like normal veins or arteries; they were large cavernous spaces
..	..	..	Necropsy	.....	.....	Mass of blood vessels in brain	Drawing of vessels suggests arteriovenous aneurysm
..	..	+ right	Necropsy	.....	.....	Mass of vessels deep in corpus striatum; extends into ventricle at caudate nucleus; connection with choroid plexus vessels	When 10 years old patient suddenly developed weakness of right arm and leg; then improved and became stationary for 20 years when paralyzed anew; 1½ years later coma and death from cerebral hemorrhage
None	None	None	Necropsy	.....	.....	Mass of small vessels in rolandic area	Patient died of cerebral hemorrhage; this lesion may or may not be an arteriovenous aneurysm; it has been described as a cavernous angioma
..	..	+ right side	.....	.....	.....	Hemorrhage into ventricle and subarachnoid space; tumor of vessels 6 by 3 cm. in frontal region	When 10 years old hemiplegia suddenly developed on the right side with speech disturbance; gradual return of function and stationary condition for 21 years when paralysis and later convulsions developed
+ localized in back	No	No	Necropsy	.....	.....	Vascular bed of vessels in right cerebellar lobe, anterior and posterior inferior cerebellar arteries run into angioma	Death from hemorrhage; much calcification and thrombosis in vessels
..	..	..	Necropsy	... .	.....	Large surface veins; collection of vessels in parietal lobe projects into ventricle	Accidental finding at necropsy following operation for sarcoma of jaw; extensive thrombosis of cortical veins even of longitudinal sinuses; dilated venous trunks are localized to region draining angioma
..	..	..	Necropsy	.....	.....	.....	This article is written in the Russian language but apparently there are two independent intracranial arteriovenous aneurysms; photographs appear to establish the lesions as arteriovenous

either petit or grand mal, since the operation (two and one-half years). The other patient, roentgenograms of whom showed the extensive calcification, was free from attacks for a year, after which they gradually recurred with practically the same intensity and frequency as before operation. The latter patient being 47 years old, the carotid was at first only partially ligated with a band of rubber tissue. Three weeks later, it was found to be totally occluded by a thrombus.

A subtemporal decompression and excision of the extracranial aneurysm (of the scalp) were performed in another patient who was losing vision because of a long-standing papilledema. His headache was relieved, but his condition otherwise was unchanged.

The cerebellar arteriovenous aneurysm had caused hydrocephalus. This was relieved by the bilateral cerebellar decompression which, as a routine, remains after a cerebellar exploration. The decompression remained full and tight until it was later relieved by ligation of the left vertebral artery in the neck. At operation, the intracranial portion of this artery was found to be several times its natural size, and this led to the belief that it supplied the arterial connection to the aneurysm. When exposed between the atlas and occipital bone, this great tortuous artery was just as large as the intracranial portion. Following its ligation, the bulging decompression subsided and has remained in this condition. Although his neurologic symptoms have greatly improved or disappeared, the mental disturbance still persists.

Ligation of the vessels, whether internal carotid or vertebral, can hardly result in a cure. Only an amelioration of the signs and symptoms can be expected, and doubtless even this cannot be permanent. The precedent for such ligations is the treatment of arteriovenous aneurysms between the carotid artery and the cavernous sinus. Following ligation of the internal carotid for these traumatic aneurysms, there is much improvement but usually not a cure. The collateral supply through the circle of Willis militates against a cure; it is fair to presume the same free collateral circulation will preclude the cure of the intracranial aneurysms.

SUMMARY AND CONCLUSIONS

1. Eight arteriovenous aneurysms of the brain are presented from a series of tumors of the brain. Twenty-two additional cases have been assembled from the literature.

2. Arteriovenous aneurysms of the brain are similar to those of the vascular system elsewhere in the body, except that traumatic arteriovenous aneurysms probably do not occur in the brain, because large arterial and venous trunks are not in apposition.

3. There are two other types of arteriovenous aneurysms, one in which an anomalous vessel of congenital origin establishes a direct end

to end communication between an artery and vein; the other in which a network of vessels—a so-called angioma—is interposed between an artery and one or several veins. A capillary bed between the artery and vein is lacking in both types; the arterial blood, therefore, passes directly into the veins.

4. Both types are evident by the large, full and tortuous veins on the surface of the brain. A thrill can usually be felt, and red arterial blood can be seen pumping into the dark venous blood. The observations make the diagnosis absolute. There are usually congenital abnormalities of the surface veins of the brain.

5. Arteriovenous aneurysms of the brain are not uncommon, occurring in about 1 per cent of a series of tumors of the brain. They are located in almost every part of the brain.

6. The symptoms are fairly uniform and characteristic. Jacksonian convulsions, followed by transient loss of sensation or motor power in the part affected by the convulsion, and a gradually progressive sensory or motor hemiplegia on the affected side, are the symptoms common to most of the aneurysms affecting the cerebral hemispheres. The symptoms are usually of many years' duration, often beginning in childhood and continuing into adult life; again, they frequently do not begin until after the thirtieth or fortieth year.

7. General pressure symptoms, like those of a tumor of the brain, do occur, but in a minority of cases.

8. Cerebral hemorrhages occurred in about 40 per cent of the cases. It is the principal cause of death.

9. Changes shown by the roentgen ray are at times helpful in making a diagnosis and occasionally are pathognomic. Ventriculography helps to determine the existence of a space-occupying lesion.

10. The treatment is of two types: (1) ligation of the entering arteries, with or without extirpation of the mass of vessels—so-called angioma; (2) ligation of the internal carotid artery (for cerebral aneurysm) or of the vertebral artery (for cerebellar aneurysm). Occasionally, a subtemporal or cerebellar decompression (depending on the location of the aneurysm) may be indicated.

Radical ligations or extirpations alone are curative, but are exceedingly dangerous to life and function and indicated in the minority of cases, when the aneurysm is posterior to the motor tracts.

LIGATION OF ARTERY AND CONCOMITANT VEIN IN OPERATIONS ON THE LARGE BLOOD VESSELS *

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The beneficial effect of occluding the satellite vein when ligation of the artery becomes necessary in surgical conditions of the large blood vessels was first recognized by Sir Arthur Makins [1] in 1913. He found a lessened incidence of gangrene in following this procedure. Nevertheless, during the early part of the World War, careful preservation of the vein was practiced, but the occurrence of gangrene following ligation of the large artery to the extremity brought forth interesting and valuable investigations on the subject. Drummond [2] showed experimentally that ligation of both artery and vein produced "little or no change" in the condition of the loops of cat's intestines. When the artery alone was occluded, definite gangrene of the segment usually appeared within twenty-four hours. Van Kend [3] attributed the decreased frequency of gangrene to a "slight rise in the blood pressure in the limb following application of a ligature to a vein, after previous ligature of the artery." He demonstrated this rise in blood pressure with plethysmographic tracings. With these and other reports presented at the Inter-Allied Conference of Surgeons in Paris, in May, 1917,[4] the conclusion was reached that "facts tend to prove, even when the wound is limited to the artery, that simultaneous occlusion of the unwounded vein is to be recommended." The subsequent application of this procedure in the surgical treatment of arteriovenous aneurysms still further strengthens its clinical advantages.[5]

Application of the principle of ligation of the vein in cases of spontaneous gangrene seems to be gaining increased favor as evidenced by its more frequent mention in the surgical literature. In most of these

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* From the Surgical Department of the Presbyterian Hospital and Rush Medical College of the University of Chicago.

1. Makins, Sir Arthur: Gunshot Injuries to the Blood Vessels, New York, William Woods & Company, 1919.

2. Drummond, Hamilton, quoted by Halsted: Johns Hopkins Hosp. Rep. **21**: 1, 1921; quoted by Makins (footnote 1).

3. Van Kend, M., quoted by Makins (footnote 1).

4. Compt. rend. Conf. Chir. Interall., Paris, 1917.

5. Halsted, W. S., Johns Hopkins Hosp. Rep. **21**:1, 1921. Holman, Emil: Ann. Surg. **85**:173 (Feb.) 1927.

cases, however, there has been a previous gradual occlusion of the main artery to the extremity. In 1913, Oppel [6] was the first to report improvement following ligation of the popliteal vein in six cases of threatened senile gangrene of the foot. He attributed the encouraging results to the retardation of the venous return flow and the consequent rise in the blood pressure of the limb. Stradin [7] also employed ligation of the vein in cases of spontaneous gangrene, reporting good results, but he combined it with sympathectomy. McWhorter [8] recently presented before his clinic at the Presbyterian Hospital, three cases of thrombo-angiitis obliterans with ligation of the femoral vein. Two of the patients had subsequent amputations, although the third showed encouraging improvement over a period of five months. As a result of experiments on animals, Brooks,[9] Holman [10] and Pease [11] have corroborative evidence for employing simultaneous ligation of the vein when the artery is occluded.

In reviewing the literature, however, I found that the final results of McNealy,[12] Thurston [13] and others, who employed ligation of the femoral vein in cases of spontaneous gangrene, were discouraging. While the value of occluding the venous return flow may not be overestimated when sudden obstruction of the artery occurs, its further use in conditions of chronic occluding diseased processes in the arteries is of doubtful value.

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6. Oppel: Zentralbl. f. Chir. **31**:1241, 1913.

7. Stradin, P.: Deutsche Ztschr. f. Chir. **194**:289 (Jan.) 1926.

8. McWhorter, Golder, L.: Personal observation and communication.

9. Brooks, Barney, and Martin, Kirley: Simultaneous Ligation of the Vein and Artery, J. A. M. A. **80**:1678 (June 9) 1923. Holman, Emil and Edwards, Muriel, New Principle in Surgery of Large Vessel; Ligation of Vein Proximal to Site of Ligation of Artery, J. A. M. A. **80**:909 (March 19) 1927. Pease, Herman: Ann. Surg. **86**:850 (Dec.) 1927. McNealy: Surg. Gynec. Obst. **40**: 45 (Jan.) 1925. Thurston, J. A.: Bull. U. S. Vet. Bur. Med. **1**:1 (Dec.) 1925. Meleney and Miller: Ann. Surg. **81**:976, 1925. Lewis and Reichert: Collateral Circulation in Thrombo-Angiitis Obliterans; Indication for Ligation of Femoral Artery First Distal to Profunda, J. A. M. A. **87**:302 (July 31) 1926. Hill, E. E.: Bull. Johns Hopkins Hosp. **35**:318, 1924. Halstead (footnote 5, first reference). Holman (footnote 5, second reference). Sehrt, E.: Med klin. **12**:1338, 1916. Stern and Cohen: Intracutaneous Salt Solution Wheal Test; Its Value in Disturbances of Circulation in Extremities, J. A. M. A. **87**:1355 (Oct. 23) 1926. Brooks and Jostes: Clinical Study of Diseases of the Circulation of the Extremities: Description of a New Method of Examination, Arch. Surg. **9**:485 (Nov.) 1924.

10. Holman (footnote 9, second reference).

11. Pease (footnote 9, third reference).

12. McNealy (footnote 9, fourth reference).

13. Thurston (footnote 9, fifth reference).

With these conflicting reports in mind, two methods were used to study the collateral circulatory bed which develops when the main arterial supply of the limb is interfered with.

1. Roentgen-ray visualization of the vascular bed in:
   (a) Amputated lower extremities in which the vessels were pathologic.
   (b) The study of the lower limbs of experimental animals following ligation of the artery alone on the one side as compared to the ligation of the artery and vein on the other side. This study was made on different animals over various periods of time up to three months after the operations.
   (c) The effect on the visualization of the collateral circulation by varying the pressure of the injection of the opaque emulsion into the diseased vessels of the amputated limbs on the one hand, and the normal vessels of the animals, on the other.
2. Determinations of the volume of blood flow were made over various periods of time on the surviving experimental animal. The determinations were made from the distal open end of the ligated femoral artery below the profunda with and without simultaneous ligation of the vein.

ROENTGEN-RAY VISUALIZATION OF THE CIRCULATORY BED

Blood Vessels in Amputated Legs.—Roentgen-ray visualization of the circulatory bed was first used by Drummond [2] in his original work on ligation of the arteries and the veins. Meleney and Miller,[14] in studying the collateral vascularity in amputated limbs for gangrene, were the first to publish its value, in a most comprehensive article. Lewis and Reichert [15] also published a similar investigation with the use of bismuth oxychloride and acacia emulsion as the material for injection instead of the previously used barium. In my experiments I find that the bismuth emulsion as recommended by Hill [16] gives the most perfect roentgenograms of the arterial tree and collaterals without diffusing into the surrounding tissues as is seen when sodium iodide solution is used. On the other hand, barium does not throw so dense a shadow as metallic mercury, and both of these are too coarse to fill the finer arterioles.

The outstanding roentgenograms of ten amputated limbs that I have studied are taken in two cases of thrombo-angiitis obliterans. The clinical histories in both cases were almost identical. In the one case, the patient was 50 years of age and of German descent. He had had symptoms of intermittent claudication, pain in the calf of the leg and discoloration and numbness of the right foot almost continuously for five years. The other patient was 48 years of age, a Russian Jew who had had similar symptoms for only two years. In both there was a complete absence of pulsation in the dorsalis pedis, posterior tibial and popliteal arteries. Even the results of the intracuticular salt solution

14. Meleney and Miller (footnote 9, sixth reference).
15. Lewis and Reichert (footnote 9, seventh reference).
16. Hill (footnote 9, eighth reference).

test [17] were exactly similar, indicating the same degree of apparent circulatory deficiency—the wheals disappearing within from five seconds on the great toe to one hour plus on the thigh. In the first case, amputation was performed because of rapidly spreading gangrene with infection; in the second, because of the extreme unremitting pain in the calf of the leg, but no gangrene. Microscopic and macroscopic examination of the arteries confirmed the diagnosis and showed almost the same degree of involvement. After injection of the popliteal artery in each case, roentgen-ray visualization revealed an extensive collateral vascular bed to account for the absence of gangrene in the one case (fig. 1), while in the other, with a much longer duration of the symptoms, an almost complete absence of the collateral vessels was found. This suggested the probability that there might be some controllable factors which could increase the formation of collateral vessels in cases of this kind.

Vascular Bed in Experimental Animals After Ligation of the Superficial Femoral Artery, With and Without Simultaneous Occlusion of the Common Iliac Vein.—Ten experiments on animal were carried out to determine the effect on the collateral circulatory bed of ligating the proximal satellite vein with the artery as compared to the ligation of the artery alone. The superficial femoral artery was ligated just below the profunda femoris on both sides, but in addition, the common iliac vein was simultaneously ligated on the right side. The animals were killed at various periods, from immediately after to three months after operation. The abdominal aorta was then injected with the bismuth oxychloride emulsion under a pressure of 125 mm. of mercury. Roentgen-ray visualization immediately after the operation showed the collateral vascular bed to be best where both the artery and the vein were ligated (fig. 2). In three weeks, however, the vascular bed was more developed where the artery alone was occluded, as compared to the opposite side where the common iliac vein was occluded simultaneously with the artery (figs. 3 and 4).

Vascular Bed During Variation in the Pressure of the Injections.—The much discussed influence of elevating the blood pressure in certain cases of circulatory deficiency due to peripheral arterial disease suggested the study of the vascular bed with variation in the pressure of injection of the extremities. Amputated limbs were first injected with a manometer pressure of around 150 mm. of mercury, the patient's own blood pressure (fig. 5). Roentgenograms were then taken and the limbs reinjected with double the pressure (300 mm. of mercury) (fig. 6). Only the faintest improvement in the finer arterioles could be detected even with such a marked increase in pressure. Further increase of pressure, however, improved the bed considerably. This probably

17. Stern and Cohen (footnote 9, ninth reference).

Fig. 1.—Roentgen-ray visualization of the vascular bed in leg amputated for thrombo-angiitis obliterans. Note the extensive collateral vascular bed with almost complete occlusion of the main arteries.

Fig. 2.—Roentgen-ray visualization with injection of the abdominal aorta under a pressure of 125 mm. of mercury immediately after operation. The collateral vascular bed is seen to be more developed where the artery and concomitant vein have been simultaneously ligated (left side of picture).

Fig. 3.—The vascular bed of the lower extremities of an animal three weeks after operation shows equal development on both sides. On the right leg the artery and vein were simultaneously occluded while on the left only the femoral artery was ligated. Note the large obturator arteries which are now the main source of arterial supply to the extremities.

Fig. 4.—Six weeks after operation the vascular bed shows greater development on the left leg where the artery alone has been ligated as compared to the right side with both artery and vein occluded.

accounts for some of the excellent roentgenograms that are frequently seen when an accurate check is not made to determine the pressure back of the injection. In order to eliminate the postmortem changes as much as possible, the limbs were studied immediately after amputation.

Considering the slight rise in blood pressure and the clinical advantage of simultaneous ligation of the satellite vein with the artery, the effect of moderate variations in the pressure of the injected material on the normal vessels of experimental animals was next studied. In order to bring out the collateral vascular bed of the nonpathologic arterial tree, the superficial femoral artery on both sides was occluded. The animal was killed, and the abdominal aorta was injected with the opaque mass under a pressure of 100 mm. and 150 mm. of mercury, and roentgenograms were made after each injection. In contrast to the slight improvement seen when the vessels were pathologic, the normal vessels showed marked improvement of the collateral bed when the pressure of the injection was increased. No doubt this difference was due to the fact that the elasticity of the artery is lost in the diseased vessel.

Determinations of the Volume of Blood Flow With and Without Ligation of the Concomitant Vein.—The temporary immediate improvement of the collateral circulatory bed with concomitant ligation of the vein as was seen in the roentgen-ray visualizations suggested the advisability of determining the volume of blood flow over a period of weeks in surviving experimental animals. This was next studied on dogs at twenty operations.

EXPERIMENTAL WORK

Large male dogs were used. Under a light ether anesthesia, the right superficial femoral artery was isolated. After a temporary distal ligature was applied, a citrated 18 gage transfusion cannula was inserted proximally into the artery. The normal flow averaged about 240 cc. per minute. The flow in each case was obtained for from five to ten seconds, and the minute determination calculated therefrom. When the cannula was removed a ligature was applied proximal to the opening in the artery, and the cannula was reinserted distally. Through an incision over the lower right rectus muscle, a previously introduced ligature around the common iliac vein was tied. The immediate flow per minute from the distal end of the ligated artery was 48 cc.; in ten minutes it was the same, in thirty minutes, 51 cc., and in one hour, 54 cc. Three weeks later, the same procedure was followed on the left femoral artery, but without occlusion of the common iliac vein. The normal proximal flow was found to be the same as on the right side. From the distal end of the ligated artery, the flow immediately following the ligation was 39 cc., increasing to 42 cc. in ten minutes, 48 cc. in thirty minutes, and 54 cc. in one hour. At the same operation, the distal end of the ligated right femoral artery of the previous operation was again isolated and cannulized. The flow was found to be 96 cc. per minute, having increased from 54 cc. during the three week interval. The distal end of the left femoral artery was recannulized at a subsequent operation three weeks later, and was found to have increased from 54 to 136 cc. per minute. All the factors, including the depth of the ether anesthesia were kept as constant as possible. No more than 50 cc. of blood was withdrawn at one operation.

Fig. 5.—Amputated limb with the injection of the opaque mass under a pressure of 150 mm. of mercury.

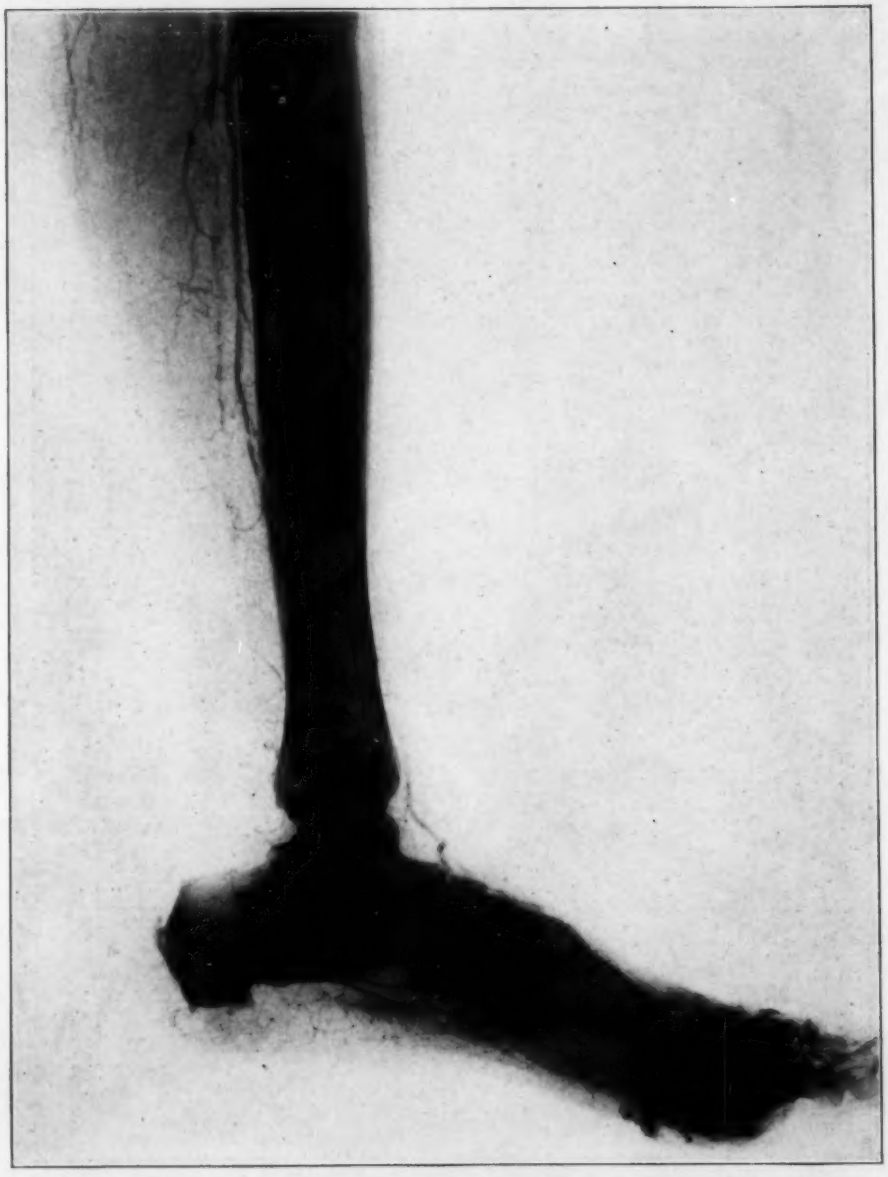

Fig. 6.—Same limb as in figure 5 with reinjection under a pressure of 300 mm. of mercury. Note that even with double the pressure only the faintest improvement in the collateral vascular bed can be detected.

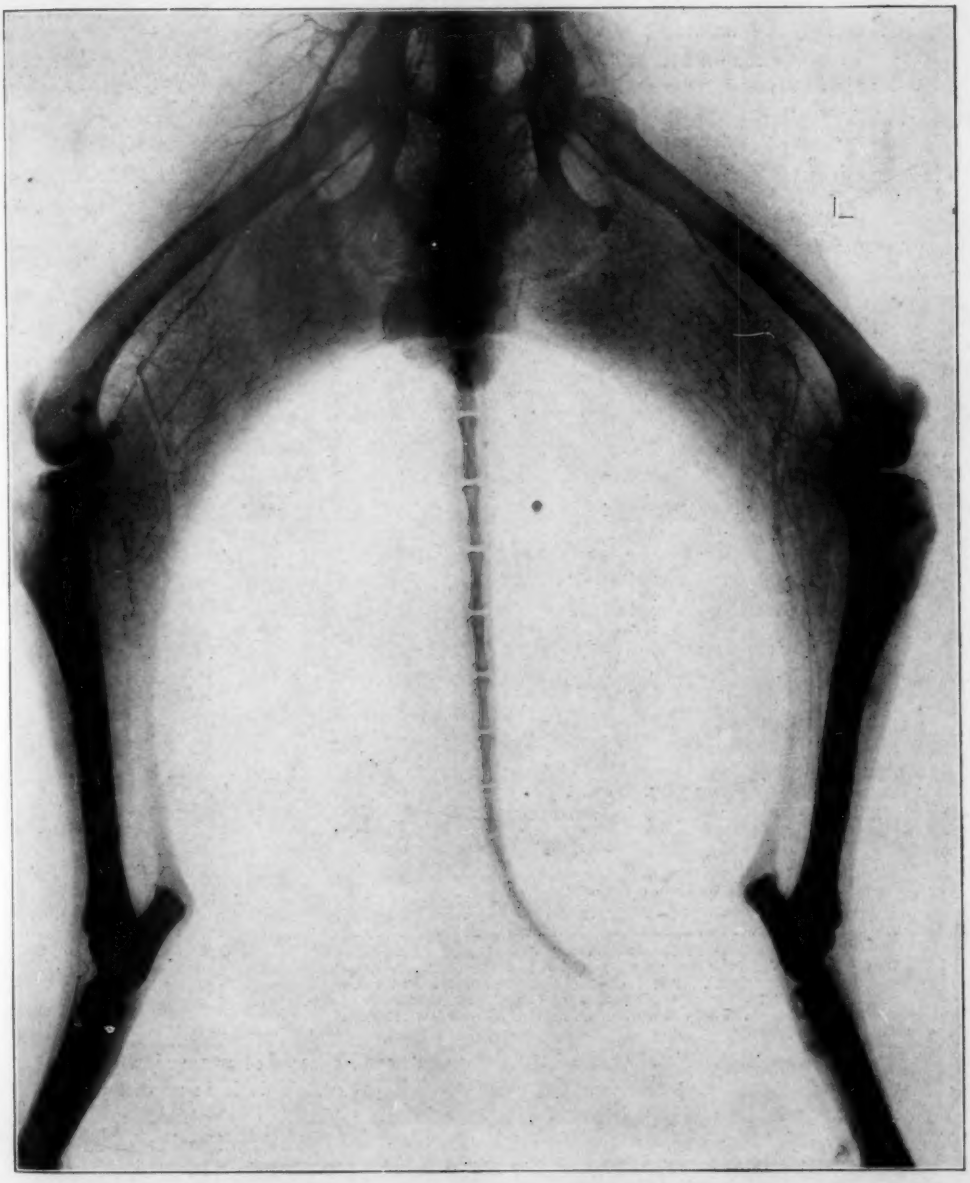

Fig. 7.—The left superficial femoral artery was ligated alone. After previous determination of the normal volume flow from the proximal end of the artery, six weeks later at subsequent operation, exactly the same flow was obtained distal to the ligature. Note in the roentgen-ray visualization the extensive development of the collateral bed which practically equals that on the right side where the circulation had not been disturbed.

Results.—Immediate transitory increase in flow from the distal end of the ligated femoral artery was obtained where the proximal concomitant vein was simultaneously occluded.

In the experiments in which the artery alone was ligated, this temporary increase was overcome and exceeded within one hour in all the animals. Moreover, when the artery alone was ligated, in one of the animals the normal flow as previously determined from the proximal end of the ligated artery returned within six weeks (fig. 7).

Determination of Blood Volume Flow in Cubic Centimeters on Dogs *

Dog	Normal	Immed. Cc.	Minutes					Weeks			
			10	20	30	40	60	2	3	4	6
I	R—	30	..	..	..	..	..	..	..	..	...
	L—	25	..	..	..	..	..	..	..	..	...
II	R—	20	..	..	..	..	..	..	..	..	...
	L—	20	..	..	..	..	..	..	..	..	...
III	R—	16	..	..	..	..	..	..	..	..	...
	L—	14	..	..	..	..	..	..	..	..	...
IV	R—	10	..	..	..	..	..	..	..	..	...
	L—	9	..	..	..	..	..	..	..	..	...
V	R—	15	..	..	..	..	..	..	..	..	...
	L—	12	..	..	..	..	..	..	..	..	...
VI	R—	14	..	..	..	..	..	..	..	..	...
	L—	12	..	..	..	..	..	..	..	..	...
VII	R—	32	..	..	..	..	..	..	.	..	...
	L—	32	..	..	..	..	..	..	..	..	...
VIII	R—	12	..	..	..	..	..	..	24	..	...
	L—	12	..	..	..	..	..	..	48	..	...
IX	R—	14	..	..	..	..	..	32	..	..	...
	L—	11	..	..	..	..	..	42	..	..	...
X	R—	22	..	..	..	..	..	..	..	..	52
	L—	20	..	..	..	..	..	..	..	..	63
XI	R—220	16	..	..	..	..	..	..	..	..	...
	L—220	12	..	..	..	..	..	..	..	..	...
XII	R—190	48	48	..	48	..	..	..	..	66	...
	L—220	42	42	..	..	54	60	..	..	90	...
XIII	R—264	80	80	90	90	..	..	..	..	..	?
	L—240	72	48	51	54	..	..	..	..	..	240
XIV	R—240	18	18	..	24	..	24	..	..	..	36
	L—240	15	18	..	21	..	24	..	..	..	42
XV	R—240	48	48	..	51	..	54	..	..	96	...
	L—252	39	42	..	48	..	54	..	..	130	...

* The left superficial femoral artery was ligated alone while on the right side the common iliac vein was simultaneously ligated with the artery. The normal flow was obtained by cannulizing the proximal end of the ligated artery, and the other determinations were made from the distal end. The flow was obtained for from five to ten seconds and the minute volume determinations calculated from the amounts obtained.

Pulsation in the distal part of the artery returned somewhat more slowly but to a final greater volume when the vein was left patent. This is in accord with the determinations of the blood flow shown in the table.

Atrophy, disability or gangrene did not develop following ligation of the superficial femoral artery, irrespective of the patency of the vein. In most cases, the circulation was decreased to below 10 per cent of normal.

COMMENT

The slight increase in the residuary arterial pressure after ligation of the artery and concomitant vein, as found by Van Kend, Brooks, Holman and Pease, must be a deciding factor in the immediate transitory improvement in the blood flow when the blood vessels are normal.

Contrary to the results reported by Holman, Brooks and others, my experiments show that the increased volume of flow with the artery and the proximal main vein ligated is only slight and transitory. After the initial improvement, there is a retardation of the volume of flow in the subsequent determinations as compared to the increase occurring on the side where the artery alone is occluded.

During the World War, ligation of the artery alone for aneurysm of the femoral artery in surgical procedures was followed by gangrene in more than 50 per cent of the cases. In experiments on dogs and rabbits, Brooks [18] reported the occurrence of gangrene in 71.5 per cent of the animals in which the artery alone was occluded and in 33.3 per cent in which both the artery and the satellite vein were ligated. In both instances, the femoral artery was ligated at the same level. Sehrt [19] reported 20 and 9 per cent of gangrene respectively, with ligation of the femoral artery, and with stimultaneous ligation of the femoral artery and vein. Irrespective of the site of the occlusion of the femoral artery, gangrene occurred less frequently when both artery and vein were occluded. Clinically, one sees gangrene develop following embolism of the popliteal artery, but not necessarily when the arterial occlusion is more gradual as in many cases of arteriosclerosis and thrombo-angiitis obliterans. Although in my series of experiments on dog, atrophy or gangrene failed to develop with occlusion of the superficial femoral artery below the profunda with or without simultaneous ligation of the common iliac vein, the ability of the collateral vascular bed to develop sufficiently rapidly is of more importance as a cause of gangrene than the point of occlusion of the artery.

The practice of ligation of the artery and the concomitant vein in traumatic surgical procedures, especially in the treatment of patients with arteriovenous aneurysm, is recognized to be of great clinical value. Experimentally, it has been impossible to confirm the enormous improvement with ligation of the artery and the proximal vein as found by Holman.[10] The difference in the results may be due to the fact that his work was done on rabbits over a short period of time and with the use of a noncoagulant in the blood. However, roentgen-ray visualization soon after the operation showed the most marked development of the vascular bed where the vein had been simultaneously occluded (fig. 2). Singularly enough, this immediate temporary benefit following simultaneous occlusion of the satellite vein in cases of sudden arterial obstruction may be the deciding factor in maintaining the vitality of the limb, the absence of which predisposes to the frequent occurrence of gangrene.

18. Brooks (footnote 9, first reference).

19. Sehrt (footnote 9, tenth reference).

With the gradual occlusion of the arterial tree that has already taken place in cases of threatened gangrene of the extremities from arterial disease, less benefit would be derived from the initial improvement following proximal ligation of the satellite vein. In these cases, the collateral vessels have had an opportunity to develop during the time that the main artery had become occluded. It is then probable that the ordinary course of events are insufficient to open the potential collaterals. The principle of ligation of a proximal satellite vein is neither applicable nor warranted as a surgical procedure in cases of spontaneous gangrene resulting from arteriosclerosis or thrombo-angiitis obliterans.

When the final maximum increase in the collateral vascular bed rather than immediate improvement is necessary, as in cases of thrombo-angiitis obliterans and other forms of threatened gangrene, ligation of the artery alone is to be recommended. Both on experimental grounds and clinically, as advocated by Lewis and Reichert,[15] this procedure is most likely to give the best results.

CONCLUSIONS

The results of the determinations of the volume of flow and the study of the injected collateral vascular bed in experimental animals show that the final increase in collateral vessels is far better when the vein is patent.

With sudden occlusion of a large artery, as in embolism or injury to the vessel, the immediate improvement in the collateral bed due to ligation of the concomitant vein is needed to maintain the vitality of the limb.

When a certain amount of collateral vessels have had an opportunity to develop, as in threatened spontaneous gangrene, surgical procedures on the main vessels of the extremities should be limited to the artery alone.

MULTIPLE RENAL STONES

PROBLEMS IN THE TREATMENT OF PATIENTS WITH THIS CONDITION

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The indications for operation in the presence of multiple stones in the kidney are not standardized and are influenced largely by the clinical data in the individual case. The tendency in recent years has been toward conservation. If in the complete removal of stones the kidney is not too greatly injured, and if it is shown that considerable function remains, the kidney should be saved. It may be comparatively easy to remove the kidney, but it requires experience and the cooperation of several technical aids to remove all the stones successfully and save the kidney. Among the factors which must be considered in determining the advisability of conservative operation are the size, shape and number of stones, the area involved, the function of the organ and the age of the patient.

SIZE AND SHAPE OF STONES

It should be recognized that the removal of large branched stones may necessitate considerable destruction of renal tissue. The removal of round, smooth stones, even if they are of considerable size, causes much less injury than removal of branched or coraliform stones which fill the pelvis and calices. Round stones are usually situated in the pelvis and seldom involve the calices; therefore their removal by pelviolithotomy is usually comparatively easy. If a branched stone involves but one calix, destruction of renal tissue resulting from its removal is not extensive. When, however, all the calices are involved, complete removal of the stones from the ends of the calices usually causes so much destruction of renal tissue that conservation is inadvisable. The complete removal of multiple small stones situated in different portions of the kidney without much injury to the renal tissue is difficult because of failure of localization. Such difficulty is greatly lessened by the aid of renal fluoroscopy. The suggestion made by W. J. Mayo and by Geraghty that large branched stones in both kidneys should be left alone unless the symptoms are so acute as to warrant emergency operation is, in general, followed. In cases of round or smooth stones in both kidneys, however, operative results are usually much better. If stones

are multiple in both kidneys, and the symptoms and shape of the stones warrant surgical intervention, every effort should be made to preserve renal tissue.

The patient's age may also be a factor in determining the operative procedure. In a case of unilateral multiple stones in an old person nephrectomy may be advisable, whereas a similar condition in a young person might warrant conservative operation. If a patient more than 60 years of age has a renal stone that is causing few or no symptoms, the stone may be left; if the patient were younger, removal would be indicated.

DIFFERENTIAL RENAL FUNCTION

The comparative estimation of function of either kidney by present methods is notoriously uncertain and misleading. Because of reflex irritation caused by the presence of stone in the renal tissue, the tests of function are frequently inaccurate. It has been our experience that reliance can be placed only on high and low values. The normal or nearly normal return of dye from the affected kidney can be accepted as an accurate index of the condition. No return of dye, or only a trace, usually indicates marked destruction of renal tissue, although in some cases a surprising amount of good renal tissue is found at operation. Intermediate values of dye return are inaccurate, and little reliance can be placed on their interpretation.

Intravenous injection with indigo carmine is the simplest functional method. The intensity of the color of the urine from either ureteral orifice affords a rough but fair index of the renal function. The differential phenolsulphonphthalein test is usually more accurate in determining the degree of reduction in function, but is equally subject to reflex inhibition of secretion and to technical error because of leakage around the ureteral catheter and faulty drainage. Nevertheless, in determining the type of operation in cases of multiple stones, especially of bilateral nephrolithiasis, differential renal functional tests may be of decisive practical value. Pyelography may be more accurate in giving an estimate of renal function than any dye test by outlining the extent and character of the pyelectasis or other deformity. In other words, extensive destruction of renal tissue is rare when the pyelogram demonstrates a comparatively normal pelvis. However, if there is evidence of marked pyelectasis, elongation and dilatation of all calices, cortical necrosis, cicatricial changes, or marked atrophy of the renal pelvis, renal injury is usually considerable, and nephrectomy may often be desirable.

RENAL FLUOROSCOPY

There is no preoperative diagnostic method which will always enable one to determine the exact number of stones present, even though shadows visible in the roentgenogram may be identified as intrarenal.

It is now generally recognized that the number of stones discovered at operation often differs from the number of shadows in the roentgenogram; therefore, a method must be employed which will ensure complete removal. Two procedures employed to accomplish this are renal fluoroscopy and roentgenograms made on the operating table. In fact, it is questionable whether a conservative operation for multiple renal stones should be attempted without using one of these methods. We have employed renal fluoroscopy in 370 cases and have found it most useful in the removal of multiple renal stones. Those who are not experienced in the use of renal fluoroscopy claim that it often fails because it does not reveal all the stones. This may occasionally be true of minute fragments from 1 to 2 mm. in diameter. It is rarely true of stones larger than from 2 to 3 mm., if correct technic is employed. Another objection may be raised because of the difficulty experienced in the exact localization of small stones, but this procedure may be greatly aided by the insertion of needles at different angles, under the guidance of the fluoroscope, so that the needle points will approximate at the site of the stone. The objection has been raised that the surgeon is placed in an awkward position when there is a small stone in the end of a calix or in the parenchyma which in spite of skill and patience he is unable to find. As a result, either considerable mutilation of the kidney may be necessary before the stone is found and removed, or it may be advisable to remove the kidney rather than to let the stone remain. There are limits to the extent of renal injury justified in searching for a fragment of stone, and it may occasionally be advisable to leave a small stone fragment rather than injure the kidney further. We have observed several cases in which a small stone was left pocketed at the end of a calix after multiple stones had been removed from the kidney, and on reexamination years later, the stone had not increased in size, had not caused symptoms, and the function of the kidney was almost normal. Leaving the stone, however, is justifiable only in selected cases of this kind. It is true that unless the kidney can be delivered above the level of the abdominal wall, it may be difficult to see it clearly, and yet if the patient is thin, it is remarkable how clearly the kidney can be seen even though not completely delivered. This difficulty has been observed in approximately 15 per cent of all cases.

No method of diagnosis is infallible, but in a review of the 370 cases of renal lithiasis in which renal fluoroscopy was employed, we have found shadows in postoperative roentgenograms in only 21 cases or 6 per cent. In most of these cases, the kidney was incompletely delivered. In several cases, the shadows were doubtful. In fact, minute shadows of no surgical significance are not infrequently observed in the roentgenogram taken soon after operation. Such shadows may not be associated with any symptoms and may either disappear in the

course of six months or may not change in a year or two. They are often not due to actual lithiasis, but to deposits of calcium in the tissues, sandy material, pieces of adhesive plaster near the external wound or bits of suture. In case small fragments remain, drainage is usually well established, and they are often passed spontaneously. In two cases in which the kidney was incompletely delivered, definite shadows of stones of 1 cm. in diameter were noted in the postoperative roentgenograms of partially delivered kidneys. It is in these cases that renal films exposed at operation as suggested by Quinby would be particularly valuable. Although this method is simpler and is more widely available, the delay in developing the films and the technical errors which may arise will retard the operation. Furthermore, localization of the stone is much less accurate.

As previously stated, the indications for operation in cases of multiple renal stones vary largely with the individual circumstances. The following cases are illustrative of many of the problems that present themselves.

REPORT OF CASES

CASE 1.—*Multiple stones in one kidney and stone in the lower portion of the ureter on the same side.*

A man, aged 27, complained of having had repeated colic referred to the left loin for several years. Results of physical examination at the clinic were negative. Urinalysis showed 35 pus cells to a field. The renal functional tests were normal. Roentgenograms disclosed the shadow of a branched stone or possibly multiple stones in the area of the left kidney; also two shadows in the region of the lower portion of the left ureter, the larger shadow measuring 1.5 by 1 cm., and the smaller 1 by 1 cm. On cystoscopy, an impassable obstruction was encountered in the left ureter at the ureterovesical juncture. The dye tests indicated moderate reduction in function of the left kidney. The right kidney was normal. The operative procedure consisted of preliminary left ureterolithotomy followed several weeks later by nephrolithotomy with removal of a branched stone and several fragments.

Comment.—If stones are present in both ureter and kidney on the same side, it is usually first advisable to remove the stone from the ureter. It has been our experience that a ureteral stone will frequently be displaced immediately following operation on the kidney and may then cause a complicating ureteral obstruction. If the stone is small and situated at the wall of the bladder, manipulation might first be tried although with stones in the kidney as well, only one or two attempts should be made because of possible complicating acute pyelonephritis. With multiple stones of this size ureterolithotomy rather than cystoscopic manipulation is indicated.

CASE 2.—*Stone in the kidney and migratory stone in the ureter on the same side.*

A woman, aged 36, had had intermittent pain referred to the right lower quadrant of the abdomen for three years. Physical examination was negative.

Urinalysis was negative save for a few pus cells. Roentgenograms showed one shadow 1 cm. in diameter in the area of the right kidney and another 1.5 cm. in diameter in the region of the right ureter at the level of the fifth lumbar vertebra. Unsuccessful attempts were made to remove the ureteral stone by manipulation. It was then decided to displace it upward by means of a ureteral catheter introduced through the cystoscope so that both stones could be removed through the same incision at operation. This was done while the patient was under anesthetic just before the incision was made over the area of the right kidney. Pelviolithotomy was then performed, with removal of both stones through the same incision.

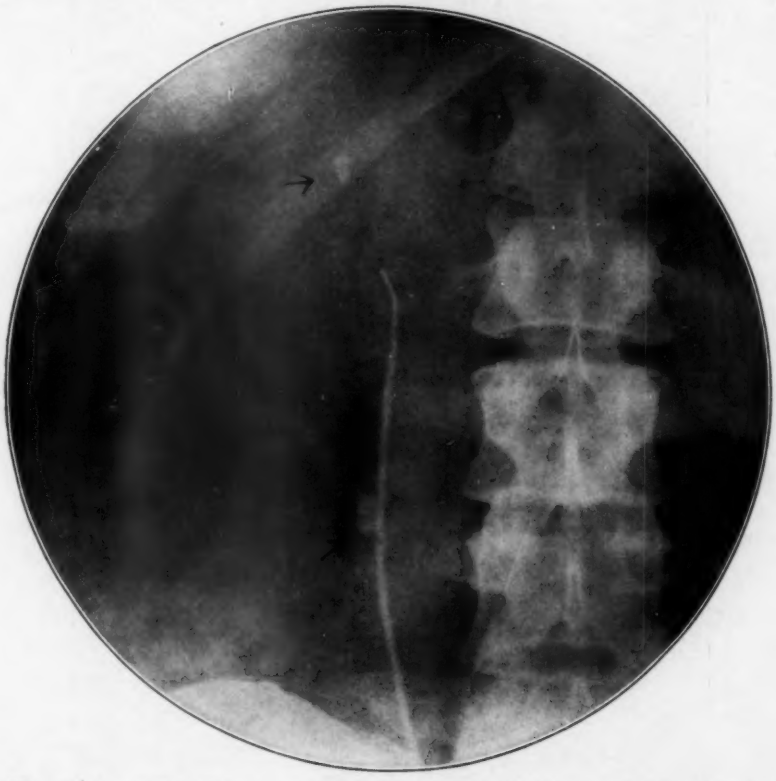

Fig. 1. (case 2).—Two stones, one in the right kidney and the other in the right ureter at the level of the third lumbar vertebra.

Comment.—The patient was saved from the ordeal of a possible second operation by this simple maneuver. It is evident that such manipulation would be possible only with a migratory stone and in the presence of a dilated ureter. Removal of a stone situated in the upper third of the ureter by means of the usual methods of ureteral dilatation is usually difficult and possible of accomplishment only after repeated trials and long delay. In the presence of two stones favorably situated, the operative procedure here employed is usually to be preferred. In

figure 1, the shadows of the two stones are visible, one in the right kidney and the other in the right ureter at the level of the third lumbar vertebra.

CASE 3.—*Stone in the kidney and pyonephrosis, and stone in the lower portion of the ureter, indicating nephro-ureterectomy.*

A man, aged 22, gave a history of recent attacks of pain referred to the upper part of the left side of the abdomen, with pyuria and hematuria. Physical examination revealed tenderness over the area of the left kidney. The phenol-

Fig. 2 (case 3).—Multiple stones in the left kidney and lower portion of the left ureter.

sulphonphthalein return was 45 per cent in two hours. The urine contained large quantities of blood and pus. On cystoscopic examination, there was evidence of marked reduction in function of the left kidney and an obstruction impassable to the catheter in the lower portion of the left ureter. Roentgenograms disclosed multiple scattered branched stones in the area of the left kidney and multiple large stones in the lower portion of the left ureter. An anterior incision was made, and the lower end of the ureter was freed and ligated. Later nephrectomy was performed through a lateral incision, and the ureter was drawn out with the kidney.

Comment.—In case of multiple stones in the kidney and ureter, and destruction of renal function, combined nephro-ureterectomy is advisable. The patient's condition may occasionally make it advisable to perform nephrectomy first and ureterolithotomy later. Large multiple stones in the ureter, if not removed, may cause considerable local infection. A widely dilated and infected ureter may act as a pocket of infection and should also be removed. If the stone is single and not larger than 1 cm., it can usually be left in situ for the time being. The majority of such stones will not cause further trouble, but should be removed if pocketing of regurgitated urine develops. Shadows of multiple stones in the left kidney and lower portion of the left ureter may be seen in figure 2.

CASE 4.—*Multiple stones in one kidney and stone in the lower portion of the ureter on the other side.*

A man, aged 32, had had repeated attacks of pain in the right lumbar area with anterior radiation during the last two years. The results of a general examination were negative. Urinalysis disclosed 50 pus cells to a field. Roentgenograms contained shadows suggestive of stone in the area of the right portion of the ureter, 1.5 by 1 cm. in diameter, probable phleboliths and shadows of multiple stones in the left kidney, the larger 2 by 2 cm. in diameter. On cystoscopic examination, the urine from both ureteral orifices was clear, and the function of both kidneys was equal and almost normal. An unsuccessful attempt was made to remove the stone from the right ureter by manipulation. At operation, right ureterolithotomy was first performed and two weeks later left pelviolithotomy.

Comment.—With stone in the ureter on one side and multiple stones in the kidney on the other side, as in this case, the indications for surgical intervention are the same as in cases of bilateral nephrolithiasis; in other words, the side which presents the most acute recent symptoms should usually be operated on first. In this case, the indication was to remove the stone in the right ureter first. Its removal by means of cystoscopic manipulation might be tried, but because of the danger of subsequent pyelonephritis and because of multiple stones in the kidney on the other side, only one or two attempts should be made. The possibility of one of the stones in the left kidney becoming displaced and obstructing the ureter with resulting anuria must always be considered. If, therefore, the acute symptoms were caused by the renal stone, nephrolithotomy should be performed first, and later manipulation of the stone in the other ureter. In figure 3, the shadow of the ureteral stone is included in the outline of the ureter which, together with the pelvis, is moderately dilated. The other shadows are shown to be extraureteral. Shadows of multiple stones are visible in the area of the left kidney.

CASE 5.—*Massive bilateral nephrolithiasis, symptomless and inoperable.*

A man, aged 20, gave a history of a slight dull ache in the right side of the abdomen for the last six months. There were no urinary symptoms. Results of physical examination were negative, except that urinalysis disclosed 40 pus cells to a field. Roentgenograms showed massive multiple stones in the areas of both kidneys and a single stone in the area of the bladder. The phenolsulphonphthalein output was 35 per cent in two hours, the blood urea was 38 mg. for each 100 cc. Cystoscopic examination showed the function of both kidneys to be slightly reduced and equal. The clinical diagnosis was bilateral nephrolithiasis with

Fig. 3 (case 4).—Stone in the ureter, extra-ureteral stones, and multiple stones in the area of the left kidney.

large branched stones filling the pelvis of the kidneys, stone in the bladder, and moderate renal insufficiency. A stone 4 by 3 cm. was removed from the bladder by litholapaxy.

Comment.—The destruction of renal tissue and the possibility of renal fistulas resulting from the removal of multiple renal stones of this type contraindicate surgical procedure unless the symptoms are so severe that life is practically unbearable. Fortunately, it is rare that large coraliform stones are the cause of acute symptoms; occasionally

a fragment breaks off and obstructs the ureter. Ureterolithotomy or cystoscopic manipulation should then be performed in selected cases. The shadows of multiple branched stones, filling the pelvis and calices of both kidneys, and of the stone in the bladder are shown in figure 4.

CASE 6.—*Bilateral stones, multiple in one kidney with single stone in the other.*

A man, aged 43, gave a history of repeated attacks of colic in the areas of both kidneys, more recently on the right side. Results of physical examination were negative, except for slight tenderness over the right kidney. Urinalysis

Fig. 4 (case 5).—Multiple branched stones filling the pelvis and calices of both kidneys, and stone in the bladder.

showed 32 pus cells to a field. The output of phenolsulphonphthalein was 20 per cent in two hours and the blood urea was 64 mg. for each 100 cc. Roentgenograms showed one large and one small shadow in the area of the right kidney and a single medium-sized shadow in the area of the left kidney. While the patient was being observed and a cystoscopy performed, the smaller stone passed from the right kidney to the lower portion of the ureter. It was necessary to perform a right ureterolithotomy at once. A right pelviolithotomy was performed several weeks later.

Comment.—In case of multiple small stones in the renal pelvis, there should not be any unnecessary delay in operating because of the danger that one of the stones may pass into the lower portion of the ureter. In such a case, if the stone cannot be removed easily by manipulation, the extra operation is indicated. It is remarkable how often the smaller renal stones will pass into the ureter while the patient is under observation, apparently influenced by the purging and manipulation incident to roentgenography and cystoscopy. Manipulation of a

Fig. 5 (case 6).—The stone over the twelfth rib on the right side moved into the lower portion of the ureter.

stone in the lower portion of the ureter in the presence of multiple stones in the kidney is more likely to be followed by acute renal infection than in uncomplicated cases. In figure 5 the smaller stone shadow, which appeared over the twelfth rib on the right side, has now moved into the lower portion of the ureter.

CASE 7.—*Bilateral nephrolithiasis: age as a factor in determining operation.*

A rather feeble man, aged 68, had had dull pain in the left lumbar area at intervals for four years. He had also experienced similar pain in the right flank on several occasions during the last year. Results of physical examination were

negative. Urinalysis showed 60 pus cells to a field. The two hour phenolsulphonphthalein output was 30 per cent, and the blood urea was 58 mg. for each 100 cc. Roentgenograms revealed bilateral nephrolithiasis and multiple round stones in both kidneys. In the left kidney, the largest shadow was 3 by 4 cm., with many small shadows. In the right kidney, the largest shadow was 2 by 3 cm.; there were multiple smaller shadows. Cystoscopic examination demonstrated that the comparative function of the two kidneys was greatly reduced, that of the left kidney being somewhat better than that of the right. It was thought inadvisable to operate on this patient because of lack of acute symptoms and also because of his age.

Fig. 6 (case 7).—Multiple bilateral renal stones.

Comment.—If the patient had been 20 or 30 years younger, operation might have been indicated, since his vitality and the life expectancy might then have justified the risk. The shape of the stones might have permitted operation. The comparatively round or smooth stones, even though multiple, would not have necessitated much destruction of tissue. Figure 6 shows shadows of multiple, bilateral renal stones.

CASE 8.—*Bilateral nephrolithiasis with one kidney practically not functioning and the other normal.*

A man, aged 25, had had attacks of severe pain in both kidneys since the age of 7 years. Recently the pain had been referred entirely to the right side. Physi-

cal examination revealed tenderness over both kidneys. The two hour output of phenolsulphonphthalein was 45 per cent. Roentgenograms showed a huge, branched stone and multiple smaller stones in the area of the left kidney and two stones, 1.5 by 1 cm. in diameter, in the area of the right kidney. The outline of the right kidney in the roentgenogram apparently indicates hypertrophy. Cystoscopic examination showed normal function of the right kidney and only slight function in the left. Right pelviolithotomy was performed, followed by left nephrectomy.

Comment.—In cases of bilateral stones of this type, in the absence of recent acute symptoms, it is usually advisable to operate first on the

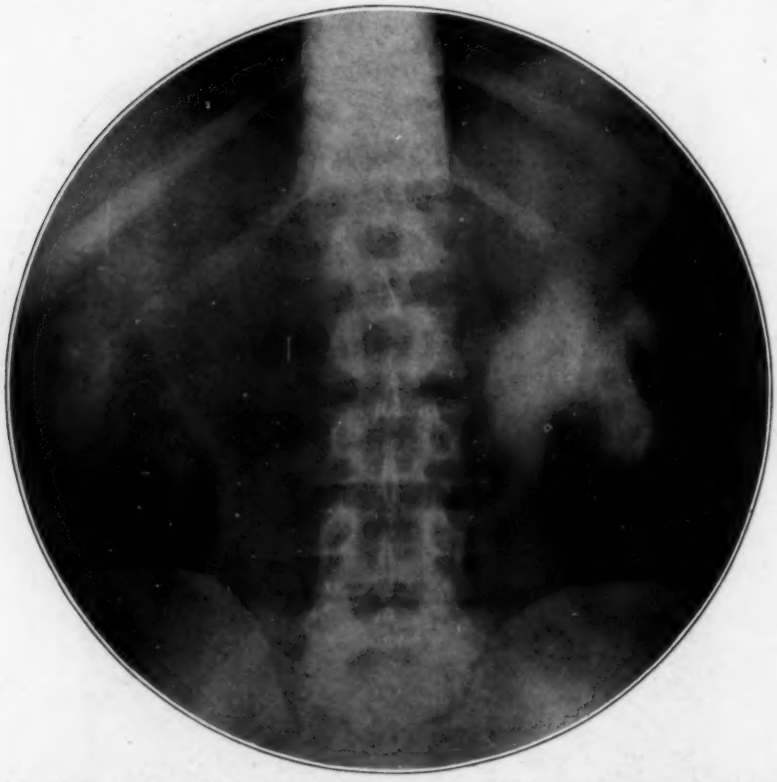

Fig. 7 (case 8).—Huge branched stone and multiple small stones in the area of the left kidney, and two small stones in the area of the right kidney.

kidney that functions better so as to take advantage of the remaining function of the other kidney. After the function of the better kidney has returned to normal, the other kidney can be more safely removed. In this case, immediate right pelviolithotomy was indicated, even though the size of the stone was favorable for spontaneous passage. Under the circumstances, the danger of ureteral obstruction by a migrating ureteral stone is obvious. In figure 7 the shadows of a huge branched stone and multiple small stones appear in the area of the left kidney, and the shadows of two small stones in the area of the right kidney.

CASE 9.—*Operable bilateral nephrolithiasis, together with stone in the ureter.*

A woman, aged 47, came to the clinic with a history of repeated attacks of colic referable to the left renal area and occasional dull pain in the right. The results of a general physical examination were negative. The roentgenograms showed shadows of multiple stones in the area of the right kidney, and one or more in the area of the left kidney, as well as a shadow 2 mm. in diameter in the area of the lower portion of the left ureter. Cystoscopic examination showed the function of both kidneys to be somewhat less than normal, the function of the right being slightly better than that of the left. A stone was seen partially protruding from the left ureteral orifice; this was cut, and it subsequently passed.

Fig. 8 (case 9).—Multiple stones in the areas of both kidneys.

Right pelviolithotomy was performed and a single staghorn stone was removed from the kidney, together with much sandy material. Subsequently, a left pelviolithotomy was done, and a stone, 2 by 2 cm. in diameter, a number of small stones and some sandy material were removed. Two months after operation, the function of both kidneys was normal and equal. Subsequent roentgenograms were negative.

Comment.—In the presence of bilateral nephrolithiasis, it is usually advisable to operate first on the kidney causing recent acute symptoms. If the symptoms are not acute, it is usually advisable to operate on the

kidney with the better function first, and, if indicated, to remove the other kidney later. Since the pain in this case was practically the same on both sides, it was deemed advisable to operate on the right kidney first because of its better function, and to remove the stone in the ureter before pelviolithotomy was attempted. As the stones were not branched and, therefore, were not wedged into the calices, they could be removed without much injury to the renal tissue. The indefinite hazy shadow adjacent to the shadows of the larger stones is explained by sandy material. Even though this is not completely removed, there is seldom danger that it will act as a nucleus for the formation of other stones if adequate drainage is established. In figure 8 may be seen the shadows of multiple stones in the areas of both kidneys. The stone in the lower portion of the left ureter is not visible in the roentgenogram.

CASE 10.—*Bilateral nephrolithiasis: multiple small stones in one kidney and a single small stone in the other.*

A woman, aged 32, gave a history of intermittent colic referred to the right loin, accompanied by pyuria, and chills and fever during a period of two and a half years. Several small stones had passed during these attacks. There were no symptoms referable to the left kidney. The results of a general physical examination were negative. The roentgenograms contained multiple scattered shadows of stones varying in size from 0.5 to 1 cm. in the area of the right kidney, and the shadow of a single stone 1 cm. in diameter in the area of the left kidney. On cystoscopic examination, the function of both kidneys was found to be normal and equal, and by means of urography the shadows were shown to be intrarenal. A diagnosis was made of bilateral nephrolithiasis, multiple small stones in the right kidney and a single stone in the left kidney. Combined nephropelviolithotomy was performed, and five cortical stones were removed from the right kidney. Fluoroscopic examination on the operating table showed that the kidney was free from stones. This observation was corroborated by a postoperative roentgenogram. The stone from the left kidney passed after an attack of colic. Cystoscopic examination one month later showed the function of both kidneys to be normal.

Comment.—It was thought advisable in this case to remove the stones from the right kidney only because the stone in the left was single, was not causing acute symptoms and was small enough to pass. The removal of multiple small stones scattered widely in the kidney may be exceedingly difficult, but with the aid of renal fluoroscopy their complete removal with a minimum of renal injury is usually possible. In figure 9 are seen the shadows of multiple small stones in the right kidney and a small single stone in the left kidney.

CASE 11.—*Multiple, bilateral stones with conservative operation indicated for both kidneys.*

A man, aged 48, gave a history of having passed stones and sand at intervals since he was 8 months old. Pain was always confined to the left side of the abdomen and had been more acute recently. The urine had been cloudy for years. The results of the physical examination were negative except that acute tender-

ness was evident over the left lumbar area. Urinalysis revealed pus in large quantities. The two hour output of phenolsulphonphthalein was 15 per cent; and the blood urea was 52 mg. for each 100 cc. Roentgenograms showed multiple shadows scattered over the areas of both kidneys. On cystoscopic examination, the urine from both kidneys was found to be cloudy, and the function of both kidneys to be greatly and equally reduced. A diagnosis was made of multiple stones in both kidneys with reduced function and infection. Eight stones were removed from the left kidney by combined nephropelviolithotomy. The kidney was three times the normal size and contained a great deal of pus. Although

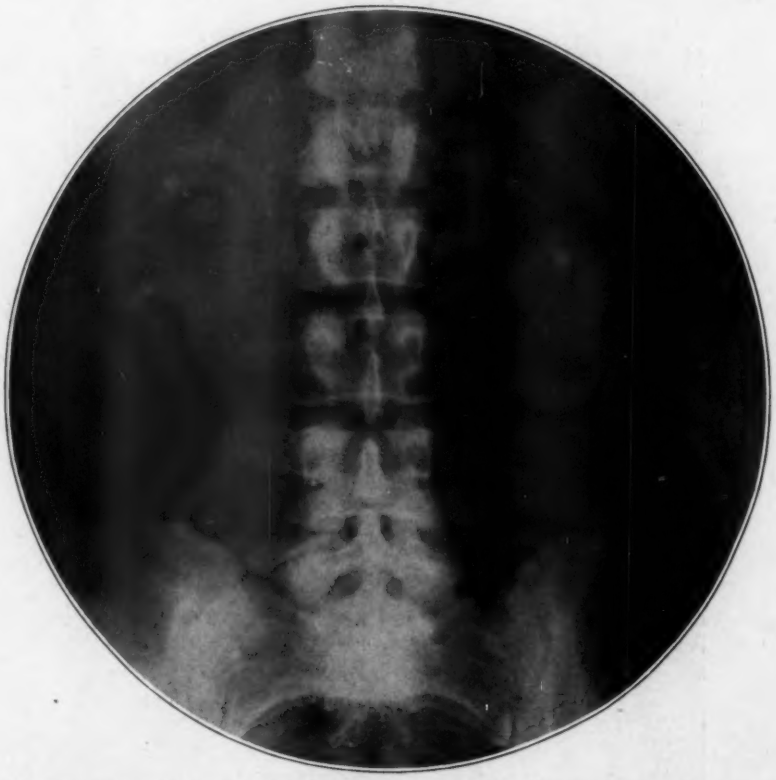

Fig. 9 (case 10).—Multiple stones in the right kidney and a single stone in the left kidney.

fluoroscopic examination showed two remaining shadows, it was considered advisable not to carry on the search since the patient was weak and the removal of the stones would have necessitated much destruction of renal tissue. Subsequently a right nephropelviolithotomy was performed and twelve stones were removed. Postoperative roentgenograms showed two fragments of stone remaining in the left renal area and none in the right.

Comment.—The case illustrates the extent to which conservative operation can be successfully carried out in bilateral multiple nephrolithiasis by employing renal fluoroscopy. It is true that two fragments of

stone remain in the left kidney, but they are in the ends of the calices, where they will probably cause little further injury. Should they increase in size, they can be more easily found and removed later The fact that twelve stones were removed from the right kidney and that a postoperative roentgenogram was negative attest to the value of fluoroscopy. Postoperative cystoscopy showed marked improvement in the function of both kidneys. The possibility for improvement in renal function following operation for multiple stones is greater when localized areas of infection are present than when they are not.

Fig. 10 (case 12).—Two large stones in both lower ureteral areas, with lead catheters adjacent to them.

CASE 12.—*Bilateral large ureteral stones.*

A man, aged 28, had had dull pain, referred to the areas of both kidneys, pyuria and intermittent spells of fever for many years. Physical examination revealed cachexia and severe enemia. Urinalysis showed a large amount of pus. The phenolsulphonphthalein return in two hours was 15 per cent, and the blood urea 44 mg. for each 100 cc. Roentgenograms showed two large shadows in the area of the bladder, which might have been caused by stones in the diverticula of the bladder or in the ureters. On cystoscopic examination, multiple diverticula

were found, but stones were not visible. A cystogram showed the outline of the stones extending beyond the outline of the bladder, so that it was evident that they were intra-ureteral. This was further corroborated by lead catheters introduced into the ureters, which were found in a urogram to be adjacent to and partially coiled around the stone. A diagnosis of two large bilateral ureteral stones and multiple diverticula of the bladder was made. At the operation, bilateral ureterolithotomy was performed, the right side being operated on first. Later the diverticula were resected.

Comment.—It is evident that any attempt at manipulation of stones of this size in a ureter is contraindicated. When complicated by diverticula in the bladder, as in this case, they should be removed before the latter are resected, because of the possibility of ureteral obstruction. Furthermore, renal function should be as nearly normal as possible before major operation on the bladder is attempted. In figure 10 the outline of two large stones is visible in both lower ureteral areas, with lead ureteral catheters adjacent to them.

CASE 13.—*Multiple stones in ureter; difficulties in removal.*

A man, aged 53, presented himself for examination in April, 1924. In 1915, a stone had been removed elsewhere from the right ureter. In 1916, three small calculi passed while the patient had colic referred to the area of the left kidney. Six months previous to the time he was examined, he experienced severe colic in the left side, and at that time roentgenograms showed a shadow opposite the third lumbar vertebra and a smaller one in the kidney. Since then a dull, dragging feeling had been noted in the left flank in the morning. Roentgenograms showed a shadow under the fourth transverse process on the left side, apparently due to multiple calculi. Urinalysis showed the presence of a few microscopic pus cells, and tests of renal function were negative. On cystoscopic examination, an impassable obstruction was met by the catheter 15 cm. above the ureteral orifice. A ureterogram showed slight dilatation of the left lower third of the ureter. There was no evidence of iodide above the shadow of the larger stone. A diagnosis was made of multiple stones in the lower third of the ureter, the largest being 2 by 2 cm., with two smaller stones just above it.

Ureterolithotomy was performed, and only one stone corresponding in size to the larger shadow could be found. The ureter was dilated above this, and it was thought possible that the smaller stones might have passed up into the kidney. A persisting fistula remained in the ureter for several weeks following the operation. Postoperative roentgenograms showed two small stones in the lower third portion of the left ureter. Several ureteral catheters were introduced into the ureter full length and fastened in it. Following repeated manipulations, both stones passed.

Comment.—With multiple stones in the ureter, and particularly if the lower one is the larger and impacted, it is logical to assume that the ureter above is greatly dilated. In such cases extra precautions should be taken not to permit the smaller stones to escape from the field of operation. With the relaxation caused by anesthesia and with the patient in the Trendelenburg position, even though the proximal portion of the ureter is partially clamped, a small stone may escape into

the upper portion of the ureter and pelvis. An attempt should be made to find the smaller stones first, since the larger stones can be picked up readily. Fortunately, in this case the stones passed following cystoscopic manipulation, although their presence caused a ureteral fistula for a period of several weeks.

CASE 14.—*Stone in kidney, ureter and bladder.*

A man, aged 38, was examined at the Clinic in February, 1925. Five years previously, he had had an attack of sharp colic referred to the upper right quadrant of the abdomen and to the lumbar area, which required several hypodermic injections for relief. Since then he had had similar attacks of pain at intervals of from two to six months. Three years previously, a stone, about 0.5 by 1 cm., had passed with hematuria. One week before he came to the clinic, the pain had been referred to the left kidney, with anuria for two days. A roentgenogram made elsewhere showed the shadows of stones in both kidneys. Recently he had complained of anuria and nausea, but he was otherwise normal. Urinalysis showed the presence of a moderate number of pus cells and red blood cells. The roentgenograms contained what appeared to be shadows of stones in the bladder, right ureter and right kidney. There was no trace of phenolsulphonphthalein in two hours; the blood urea was 282 mg. for each 100 cc., and blood creatinine was 7. A low protein diet, hot packs and forced fluids were prescribed. In three days, the blood urea dropped to 112 and the creatinine to 4.8. The diet was continued, and a week later the blood urea was 36 and the phenolsulphonphthalein return 30 per cent. On cystoscopic examination, a diagnosis was made of stone in the bladder, 2 by 1.5 cm., which had apparently recently passed from the left kidney; this explained the rapid drop in the blood urea. A urogram localized a shadow in the middle third of the right ureter, and the shadow in the kidney was included in the outline of the renal pelvis. The function of the right kidney was moderately reduced. The stone was removed from the bladder by litholapaxy. Through a right posterolateral incision, the ureteral stone situated at the middle portion of the ureter was worked back into the pelvis and removed, together with the stones in the kidney.

Comment.—The fact of greatest interest in this case was the evidence of marked renal insufficiency. In cases of multiple renal stones, the question not infrequently arises, whether operation is indicated in the presence of high blood urea retention. If there is acute ureteral obstruction, immediate operation would, of course, be advisable. In the absence of such obstruction, unless evidence of renal insufficiency can be materially eliminated by placing the patient in the hospital and prescribing a low protein diet, forced fluids and elimination, the advisability of operation is questionable. The anuria and evidence of retained nitrogenous elements in this case can be explained by the recent blockage of the left kidney by the ureteral stone, now in the bladder, and the presence of multiple stones in the right kidney and ureter. It is interesting to note the limits that the urea can attain as a result of urinary retention and then return to normal following reestablishment of urinary drainage and other treatment. In the presence of coincident stone in the bladder and kidney, litholapaxy is usually indicated, and it saves the

patient from undergoing an operation. Under such circumstances, the stone in the bladder is usually of renal origin. Also of interest in this case was the ability to work the stone in the ureter up into the pelvis so that it could be removed together with the renal stone through one incision. This, of course, is possible only when the stone is not too large and irregular and when the ureter is large enough to permit it. As seen in the urogram, the ureter was hardly large enough to permit this, but it was probably not completely distended. The pelvis and

Fig. 11 (case 15).—Two original shadows obscured by the pelvis of the left kidney.

calices may be normal in spite of the presence of a stone in the kidney and ureter.

CASE 15.—*Stones in the left half of a fused kidney.*

A man, aged 39, presented himself for examination at the clinic in July, 1924. His gallbladder and appendix had been removed at another hospital in 1921. Eight months prior to examination, he first noticed sharp pain originating in the lower left abdominal quadrant and radiating through to the back and downward into the area of the bladder. Four severe attacks during the last three months before he came to the clinic had required the administration of morphine. He had

never had any urinary symptoms. The results of a general examination were negative, including tests of renal function. Urinalysis disclosed an occasional pus cell. A renal roentgenogram contained two shadows apparently in the area of the left kidney, the larger being 1 by 1 cm. On cystoscopic examination and pyelography, an anomalous pelvis of a fused left kidney was found. The original shadows of the stones were included in the outline of the pelvis. Anomalous lateral insertion of the ureter with evidence of abnormal rotation of the pelvis was typical of a fused kidney. A subsequent pyelogram of the right side showed a similar condition. The function of both kidneys was practically normal. A diagnosis was made of multiple stones in the left pelvis of a fused kidney.

The fused kidney was revealed through an anterior incision. The pelvis of the left half of the kidney was extremely dilated, and a stone, 1.5 by 1.75 cm., was removed. The stone was shaped like a dumb-bell, giving the appearance of a second stone in the preoperative roentgenogram. Other stones could not be found on exploration.

Comment.—Multiple stones in a fused kidney offer peculiar complications in judging the necessity of surgical intervention. If the stones are large and branched, filling the pelvis of the kidney, without causing significant symptoms, an operation is hardly indicated. It must be admitted that the difficulties of operation in a fused kidney are greater than in the normal kidney. As a rule, the anterior approach with conservative operation in a fused kidney is preferable to the posterior incision. The difficulty in delivering the kidney and in completely removing the stone is necessarily greater. Unless the patient is unusually thin, it is impossible to employ renal fluoroscopy in the presence of a fused kidney, and the use of roentgen-ray films on the operating table might be applicable.

Stones shaped like dumb-bells frequently give the appearance of multiple stones. One must be certain, however, that another stone is not present, even though the shape of the stone is such that it could cast two shadows. The position of a stone of this type in itself would be suggestive of renal anomaly, since multiple stones of this kind usually are not present in the ureter. In a fused kidney, they are generally near the median line, as in this case. The appearance of the pyelogram, and particularly the insertion of the ureter and the rotation of the calices, are typical of horseshoe kidney, and it should be easily recognized.

In figure 11 the pelvis of the left kidney is visible, with two original shadows obscured by the pelvis, which is slightly dilated. Failure of complete rotation of the pelvis is typical of fused kidney. The calices are dilated.

EFFECTS OF INJECTIONS OF ACID AND TRAUMA ON JEJUNAL TRANSPLANTS TO THE STOMACH

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Experimental data are presented on further studies on intestinal segments transplanted to the stomach. Particular study has been made of the influences of the chemical and traumatic factors in the etiology of ulcer.

The almost constant observation of hydrochloric acid in clinical peptic ulcer has frequently led to the conclusion that it is an essential element in its production. Experimentally, however, it has been rather definitely established by many workers that uninjured intestinal mucosa is unaffected by exposure to the digestive action of gastric juice.

Isolated segments of intestine, with their blood supply intact, transplanted to the stomachs of dogs, remained unchanged in the experiments of Hotz [1] and Licini [2]. Later, Dragstedt and Vaughn [3] showed that various parts of the intestinal tract, kidney and spleen transplanted to the stomach remained normal when subjected to the digestive action of gastric juice for long periods. In an experimental study of transplants to different areas of the stomach, De Takats and Mann [4] obtained a small percentage of chronic ulcers in patches on the lesser curvature of the stomach near the pylorus. The percentage of chronic ulcers was even greater, when a surgical duodenal drainage was also performed (Morton [5]). The acid-alkali imbalance produced in the stomach by the latter operation and the mechanical stress to the patch on the lesser curvature were the causative factors in these ulcers. Aschoff [6] and

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4. De Takats, Geza; and Mann, F. C.: The Effect on the Jejunal Mucosa of Transplantation to the Lesser Curvature of the Stomach, Ann. Surg. **85**:698, 1927.

5. Morton, C. B.: Observations on Peptic Ulcer: IV. Patch Transplants of the Jejunum to the Stomach, Ann. Surg. **85**:879, 1927.

6. Aschoff, Ludwig: Lectures on Pathology, New York, Paul B. Hoeber, 1924.

others have emphasized the importance of the pylorus in relation to chronic ulcer in man. In Fenwick's [7] statistics from human beings 75 per cent of the chronic gastric ulcers occur in this region, while, experimentally, acute lesions frequently show a delay in healing in the pylorus. More trauma is produced in this area from food and gastric juice than in other areas of the stomach (Friedman and Hamburger,[8] Morton [9] and others).

Konjetzny [10] discussed the importance of injury to the mucosa from food and reported some interesting observations on weaned calves. Gastritis, multiple erosions and chronic ulcers were found in the stomach after the diet was changed from milk to rough food. These lesions healed when the calves were again put on a milk diet. He concluded that trauma was most important and that the gastric juice was of minor importance in the etiology of these lesions. Baggio [11] also produced chronic gastric ulcer experimentally when he fed a coarse diet mixed with 3 per cent hydrochloric acid, but he failed to produce ulcers when he fed acid alone. His experiments, however, are too few to permit of any definite conclusions.

Many types of operations have been performed for the purpose of studying the influence of acid on ulcers, by increasing the secretion of acid or prevention of neutralization by deflection of the duodenal contents into the ileum. Other methods have been the injection of quantities of acid into the stomach. Matthes,[12] in 1893, used this method in one dog and produced a delay in the healing of an acute ulcer. In investigations on monkeys and cats, Bolton [13] made a more extensive study of this method of artificial hyperacidity. He fed the animals acid solutions varying from 0.18 to 1.5 per cent. He concluded that the destructive power of gastric juice was due to the hydrochloric acid present and that the acid was usually a factor in the initiation of an ulcer.

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8. Friedman, J. C., and Hamburger, W. W.: Experimental Chronic Gastric Ulcer, J. A. M. A. **63**:380 (Aug.) 1914.

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TABLE 1.—*A Series of Jejunal Loop Transplants to Stomach*

Experiment	Date of Operation	Location of Transplant	Exploratory Operation	Interval, Days	Remarks
1	2/25/27	Posterior wall near greater curvature	Intestinal obstruction at site of anastomosis	151	Normal transplant mucosa; dilatation of duodenum
2	3/ 9/27	Middle of anterior wall	Intussusception of intestinal anastomosis	48	Transplant normal
3	4/10/27	Posterior wall near lesser curvature	Intussusception of lateral anastomosis	82	Emaciation, normal transplant mucosa
4	4/10/27	Middle of posterior wall	Transplant healed	12	Distemper developed
5	4/ 8/27	Posterior wall near greater curvature	Two chronic ulcers on ends of transplant 1.2 by 1 and 0.3 by 0.3 cm.	69	Much scar tissue at base of distal ulcer
6	4/ 9/27	Posterior wall near lesser curvature	Normal transplant mucosa	58	Marked narrowing of end to end anastomosis; some emaciation
7	4/30/27	Anterior wall, lesser curvature near pylorus	Normal transplant mucosa	72	Some emaciation
8	4/22/27	Anterior wall, midportion	Chronic ulcer on distal end of transplant 1 by 1 cm.	54	Some emaciation present
9	4/23/27	Middle of anterior wall	Normal transplant mucosa	44	Developed distemper
10	4/23/27	Anterior wall near greater curvature	Sharp spicule of bone perforating distal end of transplant	28	Serosanguinous exudate in peritoneal cavity; normal transplant mucosa
11	5/ 5/27	Anterior wall near lesser curvature	Normal transplant mucosa	48	Obstruction of duodenum due to hair ball
12	7/12/27	Middle of posterior wall	Normal mucosa	26	Some emaciation
13	7/ 2/27	Anterior wall near lesser curvature	Normal mucosa	36	Developed distemper
14	6/14/27	Middle of anterior wall	Normal mucosa	24	Developed distemper
15	7/18/27	Anterior wall near lesser curvature	Normal mucosa	22	Death from anesthesia during exploratory operation
16	7/18/27	Anterior wall near lesser curvature	Spicule of bone perforating distal loop transplant; perforation closed by pursestring suture	42	Animal died one month after exploratory operation; normal mucosa
17	8/ 1/27	Anterior wall near greater curvature	Normal mucosa	14	Condition poor after operation; loss of appetite; died

EXPERIMENTAL METHODS

Normal, healthy dogs were used. All operations were done aseptically under ether and morphine anesthesia. After recovery from the operation, the animals were given a diet of meat, bones, bread and water. The nutritional condition remained good in most of the animals until ulcers developed. The acid solutions had a tendency to decrease the animal's appetite, particularly those of a higher concentration. Variations in the amount and frequency of the injection of acid usually obviated this condition.

Openings about 8 by 4 cm. were made in different areas of the anterior or posterior walls of the stomach. Many of these openings were made close to the lesser and greater curvatures and about 4 cm. from the pyloric sphincter. A suitable length about 8 to 13 cm. of a freely movable part of the jejunum, with its blood supply maintained, was sutured into the selected areas of the stomach. The ends of the isolated segment were closed by catgut pursestring sutures. The continuity of the bowel was reestablished by end to end anastomosis, and in several by lateral anastomosis. The segment formed a part of the stomach wall with pouching of the pursestringed ends into which gastric juice and food would gravitate.

In three animals, after healing was complete, 0.62 per cent acid barium solution was injected; roentgenograms were then taken to determine the condition and position of the transplant and its effect on the emptying time of the stomach.

Exploratory operations were performed at various times from 12 to 112 days after transplantation. In five of the animals acute traumatic lesions of the transplant mucosa were made in various locations. Young's clamps were applied in one or two areas for from thirty-five to fifty minutes to produce these lesions. (In previous experiments[14] these pressures were found to produce acute penetrating ulcers of the duodenum jejunum.) In a group of seven animals, five of which had traumatic lesions, injections of hydrochloric acid were started two days after the production of the lesion of the transplant mucosa. The injections of acid were given through an Ewald stomach tube without an anesthetic for from twenty-four to fifty-eight days. In some instances the acid solution was given twice daily in amounts of from 200 to 225 cc. The concentrations of the solution were 0.22, 0.29 and 0.62 per cent. The percentages were obtained by using 3, 3.5 and 9 cc., respectively, of a 35 per cent hydrochloric acid added to sufficient water to make 550 cc. (these solutions were titrated with decinormal sodium hydroxide and found to agree with the foregoing percentages). If the solutions were injected too rapidly or in quantities of more than 225 cc., vomiting would occur immediately. This was especially true with the higher percentage of acid solution. Aspirations of the stomach from four to five hours after injection frequently gave negative results except in those animals in which a retention had been present.

Results.—Twenty-four animals with jejunal transplants were studied for a period of from 12 to 154 days. Several animals that developed an intussusception of the lateral anastomosis were not included in the group. Chronic intussusception was found in four of the group, resulting in some emaciation, but the transplant remained normal.

Seventeen animals were used for control study. Chronic ulcers occurred on the proximal and distal ends of the transplant in two, fifty-four and sixty-nine

14. Gallagher, W. J.: Acute Traumatic Ulcer of Small Intestine, Arch. Surg. **15**:689 (Nov.) 1927.

days after operation. These transplants were on the posterior and anterior wall of the stomach near the greater curvature. The mucosa was otherwise normal in these as well as the remaining seventeen control experiments. Microscopically, a moderate amount of scar tissue was present in the healed ends of the transplant. There was a marked tendency to pouch formation of the transplant. In a few of the animals, there was a dilatation of the stomach and transplant with a

Fig. 1.—Photograph taken after fixation of stomach in formaldehyde. Stomach and jejunal loop greatly dilated; loop in stomach 151 days; injections of 0.22 acid solution were made daily for 33 days; ulcers were not observed.

tendency to retention. A sharp spicule of bone was found in the distal end of the transplant in two animals. One of the spicules had perforated the transplant with a resulting serosanguinous exudate in the peritoneal cavity. A chronic ulcer, mentioned previously, was found on the end of the transplant of the other animal. In the group in which injections of acid were made, two chronic ulcers, 0.3 by 0.2

and 0.6 by 0.3 cm., were found in one animal of the seven, on the proximal and distal ends of the transplant, on the fifty-sixth day of the injections of acid and the one hundred and sixteenth day after operation. The mucosa of the transplant of this animal between the ends was hyperemic, slightly swollen and covered with mucus. This transplant was located on the posterior wall about 2 cm. from the greater curvature, near the pylorus. Edema, some hyperemia and a few petechiae

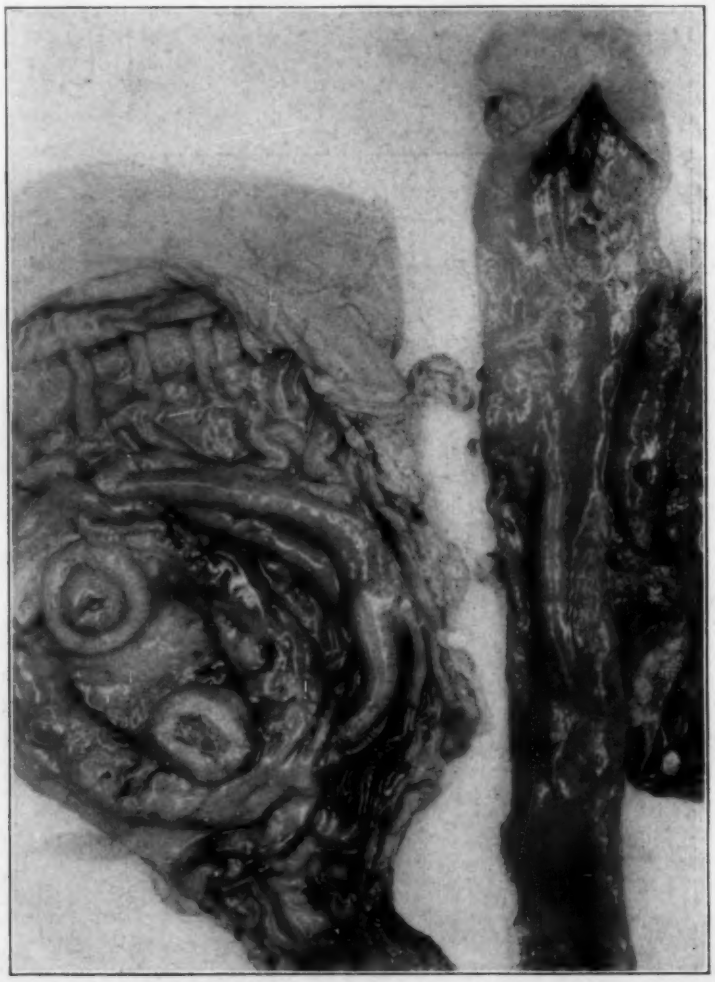

Fig. 2.—Jejunal loop with chronic ulcers on ends found sixty-nine days after transplantation to posterior wall of the stomach; lateral anastomosis of intestine, containing a fragment of bone in the free end.

were present in the mucosa of four of the animals in the group in which injections of acid were given. An appreciable amount of tenacious mucus was a constant observation in the stomachs and transplants of animals that received acid solutions. Larger amounts were found after the injection of the 0.62 per cent solution than after the use of 0.22 or 0.29 per cent acid. All the acute lesions

produced by clamps had completely healed leaving little evidence grossly of scar formation. After injection of acid for from three to four weeks, some of the animals developed symptoms of acidosis.

Microscopically, there was a moderate amount of cellular exudate in the mucosa of the transplant of the group which received injections of acid. Goblet cells were found in the mucosa of a few of the transplants.

COMMENT

Scar tissue was constantly present in the ends of the transplants, in which chronic ulcers were found in three animals. Consequently, the most important etiologic factor was undoubtedly the nutritional dis-

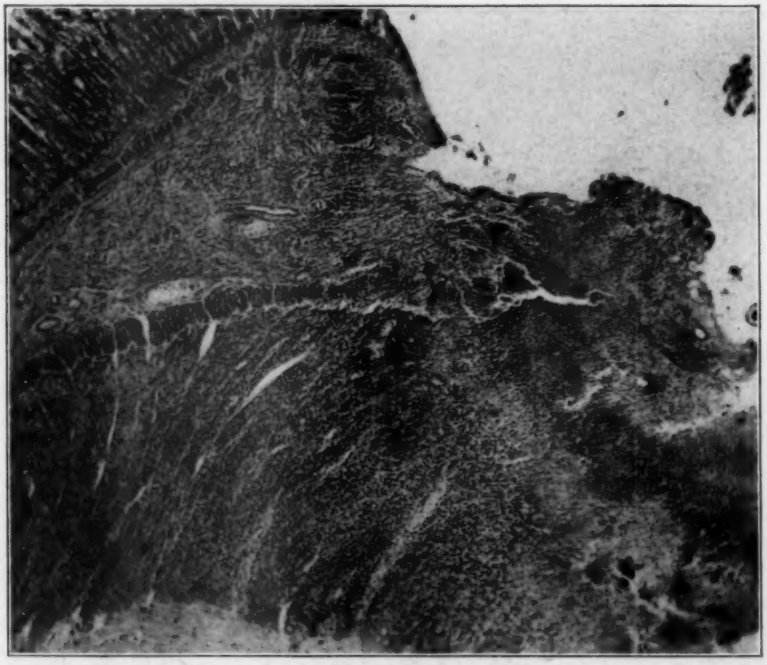

Fig. 3.—Photomicrograph of chronic ulcer on distal end of transplant shown in figure 2; superficial necrosis with abundant scar tissue in base of ulcer; × 32.

turbance present in these regions of the transplant. The location of the segment in the stomach seemed to have little influence on the occurrence of ulcer in these experiments. In those transplants with chronic lesions, the distal lesion was always larger than the proximal one. This is logical, since the distal part was more frequently subjected to trauma from food and perhaps the action of gastric juice.

Several times at exploratory operation and postmortem examination, the distal end of the transplant contained food, bones and gastric juice, while the proximal end was usually empty. The tendency to retention in this portion of the transplant was also observed in the roentgen-ray

TABLE 2.—*A Series of Injections of Hydrochloric Acid Into Stomachs with Jejunal Transplants (Acute Traumatic Lesions Produced by Clamp Pressure)*

Experiment	Date of Operation	Location of Transplant	Injection of Hydrochloric Acid			Acute Traumatic Lesion		Interval, Days	Necropsy Results	Remarks
			Percentage	Amount Injected, Cc.	Number of Days	Date of Operation	Location of Transplant			
18	2/12/27	Posterior wall, middle	0.22	200	33	5/11/27	Distal third	151	Dilated stomach, much mucus present; hyperemia of mucosa of transplant; traumatic lesion healed	Edema of lungs from aspiration of acid solution
19	4/13/27	Posterior wall, near greater curvature	0.29	200	56	.....	.....	116	Edema of transplant mucosa; two chronic ulcers on ends of transplant, 0.3 by 0.2 and 0.6 by 0.3 cm.; mucus in stomach	Frequent vomiting of mucus and acid solution
20	4/15/27	Middle of posterior wall	0.29 0.62	200 200 twice daily	53 29	8/ 5/27	Middle of transplant	154	Edema and petechiae of transplant mucosa; mucus in stomach and transplant; traumatic lesion healed	Developed symptoms of acidosis
21	6/11/27	Middle of anterior wall	0.22	200	32	.....	.....	56	Mucus in stomach and transplant; mucosa normal	Frequent vomiting of acid solution
22	7/16/27	Posterior wall near lesser curvature	0.62	200	60	8/10/27	Two areas, middle and lower third	78	Traumatic lesion healed; much mucus present; some edema of transplant mucosa	Slight cellular exudate in mucosa of transplant
23	7/27/27	Anterior wall near greater curvature	0.62 0.62	200 200 twice daily	27 32	8/ 8/27	Middle	73	Healed traumatic lesion; some edema of transplant mucosa; much mucus present	Developed symptoms of acidosis
24	8/ 2/27	Anterior wall near lesser curvature	0.62 0.62	200 200 twice daily	..	8/16/27	Two areas, middle and proximal portions	41	Edema and petechiae of mucosa transplant; traumatic lesion healed; much mucus present in stomach and transplant	Slight cellular exudate in mucosa of transplant; developed symptoms of acidosis

examination, when barium material was observed after the upper part of the transplant was empty. The tendency of the transplant to pouch formation protected the mucosa from the greater trauma from food and the action of gastric juice. It was also a factor in aiding the healing of acute traumatic lesions. Observations were not made on the time of healing of these lesions. Grossly and microscopically the healed lesions were not unlike those found in the duodenum and jejunum in previous experiments. Bolton [13] reported a delayed healing of acute ulcers in the stomach following the injection of solutions of hydrochloric acid.

The constant presence of gastric mucus in the experiments with artificial hyperacidity was, no doubt, with the alkaline secretion of the intestine, a protective influence against the irritant action of the hydrochloric acid. Frequently vomiting of acid solutions, especially those of higher concentration, also decreased the efficiency of this method of hyperacidity. The rapid emptying of the stomach of acid solution (Carlson,[15] Steinberg,[16] Olch [17] and others) still further limited the time of action of the acid on the transplant mucosa. The few changes observed (hyperemia and edema) are in part explained by these factors. The fact that the higher concentration of acid (0.62 per cent) did not produce any greater changes in the transplant mucosa than the lesser concentration solution (0.29 per cent) is further evidence of the protective influence of the various factors mentioned.

In these experiments, trauma, due to operation, and lessened blood supply to the ends of the transplant caused nutritional disturbances which determined the site of the mucosal ulceration.

SUMMARY

Chronic ulcers were observed on the ends of two transplants of seventeen animals, in which the blood supply to the loop was maintained. These transplants were on the posterior and anterior wall of the stomach near the greater curvature. The longest period of observation was 154 days.

An artificial hyperacidity was produced by injections of varying concentrations of hydrochloric acid from once to twice daily into the stomach in seven animals, for periods of from twenty-four to fifty-eight days. In one of these, two chronic ulcers were observed similar to

15. Carlson, A. J.: Personal communication to the author.

16. Steinberg, M. E.; Brougher, J. C., and Vidgoff, I. J.: Changes in the Chemistry of the Contents of the Stomach Following Gastric Operations, Arch. Surg. **15**:749 (Nov.) 1927.

17. Olch, I. Y.: Duodenal Regurgitation as a Factor in Neutralization of Gastric Acidity, Arch. Surg. **16**:125 (Jan.) 1928.

those in the control experiments, 112 days after operation and 58 days after the injection of 0.62 per cent hydrochloric acid. In four animals of this group, edema and some hyperemia of the mucosa were noted.

Gastric mucus was a constant observation in the stomachs and transplants of the series in which injections of acid were made. Acute traumatic ulcers of the transplants in six of the series with artificial hyperacidity healed with only slight gross changes. Nutritional disturbance of the ends of the transplants was suggested as the probable important factor to explain the presence of chronic ulcers found in these experiments. The gastric mucus and alkaline secretion of the intestine were considered an important protective influence to the mucosa against the irritant action of the hydrochloric acid solution injected.

CONCLUSION

In these experiments the attempt at artificial hyperacidity failed to increase the occurrence of chronic ulcer in the stomach itself, in the scar tissue of the transplant or in the transplant mucosa.

PHYSIOLOGY OF THE LIVER AND INTESTINE

METHOD OF STUDY *

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BALTIMORE

I shall make a preliminary report on a method for the study of the function of the intestine and liver, with particular reference to the study of the portal blood. This method is for experimental use in animals.

An instrument or permanent cannula is used between the portal vein and the skin of the abdominal wall. It is a gold plated cannula having two tubes.

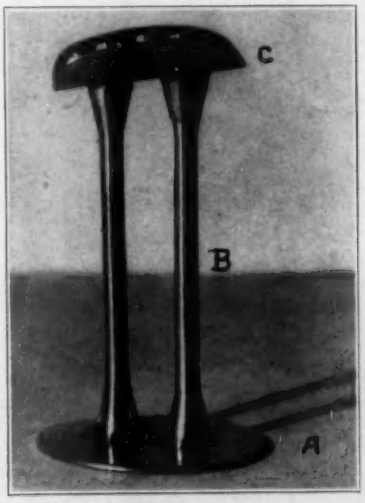

Fig. 1.—Instrument used in experiment.

These tubes barely allow the passage of an 18 gage record needle. Sterling silver tubing is used because it is impossible to plate the lumen of the tube.

The instrument consists of two tubes *(B)* connecting a concave portion *(C)* which is sutured over the portal vein to a round flat plate *(A)*. The round flat plate is placed beneath the skin of the abdominal wall. It is about the size of a half silver dollar (fig. 1).

The operation is performed with the animal under general anesthesia. A half moon incision is made in the upper right quadrant. The convex surface of the incision points toward the midline. A longitudinal incision is made through the rectus muscle and peritoneum. The portal vein is exposed, and

* From the Department of Surgery, University of Maryland.

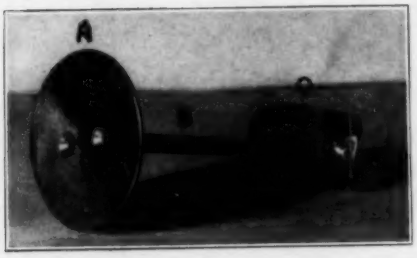

Fig. 2.—Depression in the plate (*A*) for the entrance of the needle into the tubes.

Fig 3.—Dog two weeks after operation. The arrow points to the instrument.

the curved plate of the instrument (*C*) is sutured over the portal vein at the junction of the splenic vein. A curved incision is used to prevent the round plate of the instrument (*A*) from being placed beneath the scar of the incision. That portion (*A*) is therefore lateral to the incision. This can be seen in figure 3.

The illustration in figure 4 shows how the instrument is sutured over the portal vein. Four sutures are necessary to hold the instrument in place. It is better to apply all sutures before tying the knots.

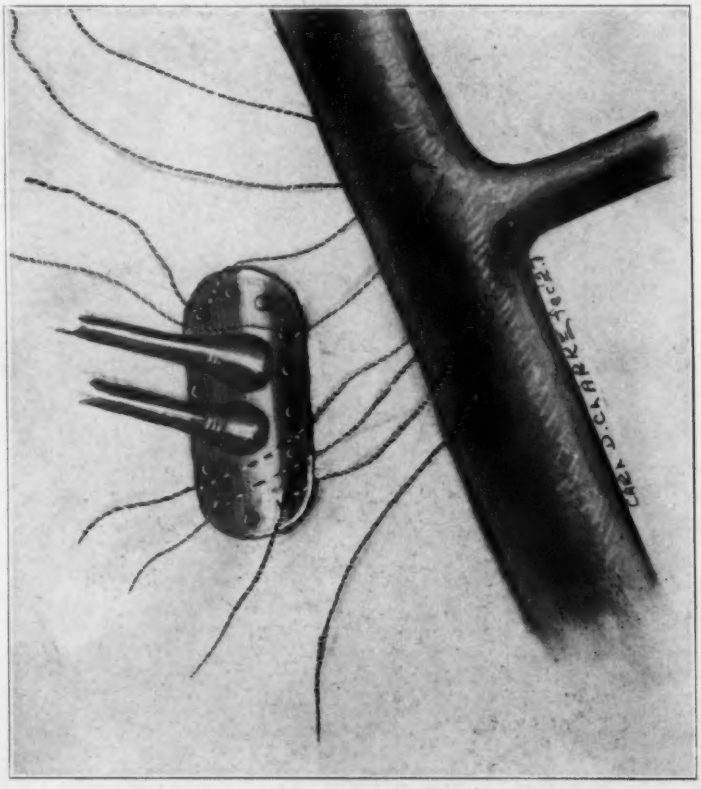

Fig. 4.—Illustration showing how instrument is sutured over the portal vein.

In figure 6 the instrument has been sutured over the vein. This has been done through the incision of the rectus muscle. The semicircular flap incision in the skin, and the plate (*A*) resting on the lower ribs should be noted.

The plate (*A*) is brought through the abdominal wall by making a second incision along the costal margin near the lower ribs. This incision includes the muscles and is not made through the skin. The instrument is suspended on the lower ribs to prevent pressure on the vein. The wound in the rectus muscle and peritoneum is closed; catgut is used in the peritoneum and silk in the sheath of the rectus muscle. The semicircular skin incision is sutured with interrupted sutures of silver wire.

A great many dogs were killed because of infections in the wound. This was overcome by placing a silver wire drain (made of twisted strands of silver wire) in the wound, for a period of from two to three days. Otherwise the dogs would develop a collection of serum beneath the skin with a secondary infection of the wounds. The wounds usually heal in from ten days to two weeks, and the vein may then be aspirated.

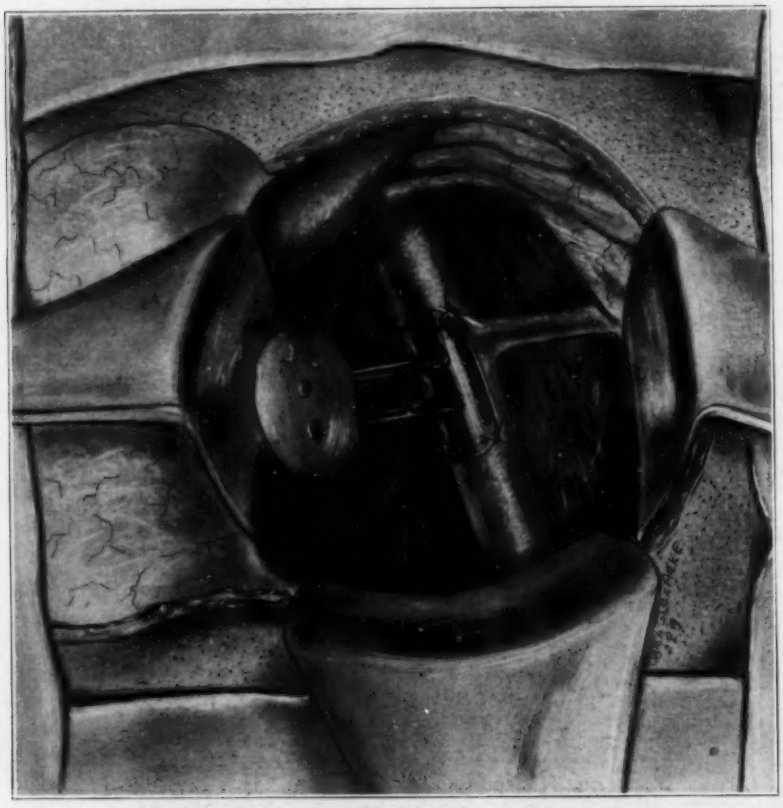

Fig. 5.—Instrument after it is sutured over the vein.

ASPIRATION OF THE BLOOD

The round plate of the cannula is grasped between the thumb and index finger. An 18 gage Record needle is passed through the skin into the depressed surface of the plate (*A*). The needle should be attached to a Record syringe and immediately after it is passed into the cannula, suction should be made with the piston of the syringe. If this technic is used, the portal blood will be aspirated into the syringe on the immediate entrance of the needle into the vein. After the desired quantity of blood is obtained, the needle is withdrawn from the vein, a distance of about one-fourth inch, and a stylet is placed in

Fig. 6.—Instrument sutured over the vein by means of incision of the rectus muscle.

Fig. 7.—Close up view of the abdomen of the dog shown in figure 3. The scar of the semicircular skin incision and the plate of the instrument beneath the skin, as indicated by the arrow, should be noted.

Fig. 8.—A dog eighteen months after the introduction of the cannula. Blood may still be aspirated from the portal vein.

Fig. 9.—Close up view of the abdomen of the dog shown in figure 8. The arrow points to the instrument.

the needle. This controls hemorrhage from the punctured wound of the vein. The needle is allowed to remain in this position for a few minutes and then may be withdrawn.

Several points should be emphasized in the application of this method besides those already mentioned. Cannulas of various sizes should be made that may be applicable to dogs of different sizes. It would be difficult to use an adjustable instrument. The instrument should be of sufficient length so that there will be neither pressure nor too much tension on the vein. The portal vein in the dog is freely movable, and too much tension by suspension of the instrument on the rib as already described is not likely to occur. If the needle is introduced as described and a stylet placed in the needle after aspirating the blood, hemorrhage will not occur. It is true that the use of a small needle may give rise to some difficulty by the formation of a clot in the needle. If this should occur, the needle may be withdrawn and cleansed or another needle may be used. It is possible that the needle may pass through the portal vein and enter the inferior vena cava. I am now using another separate plate directly over the vena cava, like the plate *(C)* of the instrument. This plate makes it impossible for the aspirating needle to penetrate the vena cava.

The method has been used successfully in ten dogs. Unlimited aspirations may be made. Figures 8 and 9 show a dog that has retained the instrument for eighteen months. The portal vein in this dog has been aspirated more than fifty times. The dog apparently is normal and has gained weight since the introduction of the cannula. In three of the dogs, the round plate of the cannula ulcerated through the skin in from six to eight months after the introduction of the cannula.

The cannula may be used in the study of absorption from the intestinal tract, in tests on the function of the liver and in the study of the various dyes in tests on the function of the liver by the introduction of these dyes directly into the portal vein.

TOXIC GOITER AND MENTAL DISEASE

RELIEF OF PHYCHOSES IN THYROTOXIC PATIENTS BY THYROIDECTOMY *

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CINCINNATI

It has long been recognized that disturbances of the thyroid gland may give rise to mental disease, but hitherto attention has been focused largely on ablation of the thyroid function as the principal cause of thyrogenic psychoses. Before thyroid extract was employed, many patients with myxedema were confined in insane asylums. Without treatment, this disease may give rise to so extreme a degree of psychic paralysis that institutional care becomes necessary. Memory is lost, power of volition disappears, and the patient becomes completely indifferent to his surroundings. In cretinism, a somewhat similar train of mental symptoms follows.

The relation between toxic goiter and mental alienation is less generally admitted, yet I have made some observations that have led me to believe that under certain circumstances thyrotoxicosis may be the exciting, or at least a strong contributory, cause of serious mental disease, and that in such cases, particularly when the psychosis is not far advanced, operation may offer a good prospect of mental recovery. During the past five years I have performed thyroidectomy on fourteen frankly insane patients with toxic goiter; all but two recovered sufficiently to resume their former occupations.

LITERATURE

In 1908, Berkley and Follis [1] reported a study of a series of cases of the catatonic form of dementia praecox, in which they found a definite resemblance between symptoms of exophthalmic goiter and those of catatonia: chiefly, rapid pulse, muscular tremor, hyperhidrosis, overactive eye and other reflexes, rapid loss of weight, disturbance of the menses and vasomotor pareses. They observed that the psychic changes in the prodromal stage of catatonia also resemble those seen in exophthalmic goiter. In most of their cases of catatonia, the thyroid gland was found to be soft and mushy with occasional hard nodules. Usually it was not enlarged. Experiments showed that the administration of iodine, thyroid extract or desiccated thyroid woke the patients out of

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* From the Department of Surgery, DeCourcy Clinic.

1. Berkley, H. J., and Follis, R.: An Investigation into the Merits of Thyroidectomy and Thyro-Lecithin in the Treatment of Catatonia, Am. Med.-Psychol. Assoc. **15**:283, 1908.

their stupor, so that they became talkative and excited and exhibited increased dermographia and hyperhidrosis, higher pulse rate and increased tendon reflexes.

A transitory amelioration of the mental symptoms has frequently been observed in cases of dementia praecox following thyroid medication, but lasting benefit has rarely been reported. Wagner-Jauregg,[2] however, noted a favorable influence from treatment with thyroid in some early cases of the hebephrenic type.

Berkeley and Follis performed partial ablation of the thyroid gland in ten cases of catatonia. Four of the patients recovered their mental equilibrium after operation; three improved slowly but progressively without a tendency to relapse. In one of these cases, the pathologic appearance of the thyroid gland was suggestive of exophthalmic goiter. The fourth patient improved rapidly for a time, but relapsed for a few months, during which period the remaining half of the thyroid gland increased in size. When this goiter again decreased in size, the mental condition became normal and remained so. In this case, no pathologic condition other than colloid goiter was found in the portion of the gland that was removed. In another case, there was less improvement but mental deterioration was more advanced, and only a small portion of the gland was removed. In four cases, there was slight fibrosis. Two of the patients were cured by the operation.

Kanavel[3] reported the results of thyroidectomy in eleven cases of dementia praecox, eight of them of the catatonic type. All but two represented cases of from two to ten years' duration. Improvement in the mental condition was not noted after operation. Of the two recent cases of less than one year's duration, one occurred in a patient who had been operated on too recently to judge of the result. The other patient showed definite improvement, but not complete cure. Another case, not included in this series, was noted, in which the thyroid gland was removed because of symptoms of pressure due to enlargement. The operation resulted in marked mental improvement, so that the patient was discharged from institutional care and returned to work. In the eleven cases, ten of the thyroid glands removed were examined pathologically. Only two showed excessive parenchymatous growth; six showed colloid changes. Kanavel noted, however, that colloid glands may result from a degenerative change, and this observation does not absolutely exclude the assumption of a previously perverted excessive thyroidism. Eight of ten patients in this series showed changes in the

2. Wagner-Jauregg: Organotherapie bei Neurosen und Psychosen, Wien. klin. Wchnschr. **36**:1, 1923.

3. Kanavel, A. B.: The Advisability of Thyroidectomy in Catatonic Dementia Praecox, Illinois M. J. **16**:253, 1909.

thyroid gland compatible with the assumption of a thyroid origin of the mental disease. Morse,[4] on the contrary, failed to find any uniform lesions in the thyroid gland in a series of twenty-seven postmortem examinations on patients with dementia praecox.

In 1910, Winslow[5] reviewed the results obtained by Berkley and Follis and by Kanavel and reported five additional cases of dementia praecox in which partial thyroidectomy was performed. One of these patients died with acute thyroidism; two were greatly improved for a considerable period and then relapsed; one was not materially benefited; and one was improved, but could not be kept under observation. From these cases and the results reported by others, Winslow concluded that there seems to be some connection between the thyroid gland and catatonia; that partial thyroidectomy in the early stages of this disease has a favorable, and sometimes a curative, effect; but that, in the late stages, the operation does not have any beneficial results.

In 1922, Weinberg[6] reported eight cases of mental disease with symptoms of hyperthyroidism and somewhat enlarged thyroid glands, but not the typical symptoms of exophthalmic goiter. Two of the patients had the manic-depressive type of psychosis; three, dementia praecox. Thyroidectomy was performed in three cases of this series, including one of the manic-depressive and one of the dementia praecox type, and resulted in complete relief from the mental symptoms.

In 1922, Eastman and Eastman[7] stated:

> Since all the clinical symptoms of Graves' disease are largely of nervous origin and all are emotional, many cases exhibiting rather definite forms of nervous and mental disease as mania, chorea, hysteria, etc., it is not at all surprising that the rather definite clinical syndrome of dementia precox appears occasionally in connection with thyrotoxicosis.

They reported that at the Central Insane Hospital at Indianapolis, 101 consecutive patients with dementia praecox of all types were examined for signs of endocrine imbalance, and especially for thyroid hypertrophy. Of twenty-seven male patients, eleven, or 40 per cent, showed some degree of thyroid enlargement. Two of these patients had large goiters; six, a conspicuous enlargement of the gland; three, moderate hypertrophy of one or both lobes. Of seventy-four female patients, forty-eight, or 64 per cent, showed some thyroid hypertrophy. Eighteen

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4. Morse, M. E.: Pathologic Anatomy of Ductless Glands in Dementia Praecox, J. Neurol. & Psychopath. **4**:1, 1923.

5. Winslow, R.: Partial Thyroidectomy in Dementia Praecox, J. A. M. A. **55**:1195 (Oct. 1) 1910.

6. Weinberg, H. M.: Mental Side of Hyperthyroidism, Penn. M. J. **25**:618, 1922.

7. Eastman, J. R., and Eastman, N.: Thyroid Surgery and Dementia Praecox Syndrome, Ann. Surg. **76**:438, 1922.

of these had actual goiters, nine of which were apparently cystic. In both male and female patients, however, "the cardinal symptoms of thyrotoxicosis, except goiter, were notably absent." Eastman and Eastman reported four cases, all in females with the paranoid type of dementia praecox, in which one lobe of the thyroid or one lobe with the isthmus was enlarged. Two of the patients showed slight exophthalmos; the other two did not show any ocular signs of exophthalmic goiter. There were no frank symptoms of thyrotoxicosis, but all patients showed accelerated pulse rate and some tremor of the fingers. A hemithyroidectomy was performed in the four cases and in all the psychosis was entirely relieved, the patients returning to their usual occupations.

In 1924, Jackson [8] reported a psychosis in a woman, aged 53, who had typical symptoms of exophthalmic goiter, although exophthalmos was slight. The thyroid gland was symmetrically enlarged to a slight degree. It felt firm and fibrous, being a typical hyperplastic gland. The right superior thyroid artery was first ligated. This operation resulted in a severe reaction with exacerbation of all the symptoms. Ligation of the left superior thyroid artery, however, resulted in marked improvement. Later, two thirds of the right lobe and isthmus were resected. Two weeks after this operation the basal metabolic rate fell to normal, and the patient was mentally normal.

In 1926, Boys [9] stated that in his series of cases of toxic goiter he found that about 2 per cent show mental symptoms so severe that they may be called definite psychoses. He reported eight cases in which there were well developed mental symptoms and goiter. In six of these cases, removal of the thyroid gland resulted in complete recovery. The thyroid glands were grossly or microscopically hyperplastic, and the symptoms of hyperthyroidism had preceded the development of the psychosis. In one case there was an adenomatous goiter without hyperplasia, and in another, a simple colloid goiter. In neither of the cases were the mental symptoms improved by operation, nor had the symptoms of goiter preceded the development of the psychosis. One of the patients relieved by thyroidectomy was affected with the manic-depressive type of psychosis.

In 1924, Foss and Jackson [10] reported an incidence of 59 cases of goiter among 1,647 patients at the State Hospital in Danville, Pa. As this institution is in a goiter belt, this is not a high incidence of goiter. Forty-nine of the goiters were in women; ten in men. In both

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8. Jackson, A. S.: Value of Ligation in Psychosis of Exophthalmic Goiter, Wisconsin M. J. **22**:426, 1924.

9. Boys, C. E.: Psychoses Associated with Goitre, J. Michigan M. Soc. **25**: 653, 1926.

10. Foss, H. L., and Jackson, J. A.: Relationship of Goiter to Mental Disorders, Am. J. M. Sc. **167**:724, 1924.

sexes, goiter was most frequently associated with manic-depressive psychosis and dementia praecox. The goiters were chiefly of the adenomatous type; a few, of the colloid type. There were no goiters of the hyperplastic type, and evidence of hyperthyroidism was not present in any case. In a few cases the thyroid was removed, but without effect on the psychosis. In all instances, typical adenomatous changes were found in the removed gland. There were no cases of definite hyperthyroidism. In a study of 800 patients with goiter in a general hospital in the same locality, Foss and Jackson did not find any cases of true insanity. Only two patients showed mild excitement or slight and transient mania. Foss and Jackson, therefore, did not see any definite relationship between goiter and insanity, but they admit that thyroidectomy should be performed when there is evidence of hyperthyroidism in a person with a psychosis.

In 1923, Weiss [11] reported seven cases of psychoneurosis with anxiety states associated with definite symptoms of hyperthyroidism; that is, tachycardia tremor, glittering eyes and moist hands. Partial thyroidectomy in two cases, and roentgen-ray treatment of the thyroid gland in one case, failed to relieve the patient from the psychosis. Weiss believed that the thyrotoxicosis in these cases was not directly responsible for the mental symptoms, but that the two developed concomitantly, both as symptoms of an abnormal degenerative constitution, most probably with disturbance of the vegetative nervous system.

Studies of basal metabolism in dementia praecox indicate that a lowered rate is more frequent than high basal metabolism, which is characteristic of hyperthyroidism. In 1925, in a study of the basal metabolism in 161 cases of mental diseases of various types, Bowman and Fry [12] found that of twenty-four patients with dementia praecox, eighteen showed minus readings, which were abnormally low in seven instances. The manic-depressive group showed a tendency toward low readings in the depressive and hypomanic stages, but this was not so marked as in the cases of dementia praecox. In a review of the literature on basal metabolism in dementia praecox, Bowman and Fry found 125 cases (including their 24 cases). The basal metabolism was high in only eleven of the cases; in fifty-two, it was low, and in fifty-two, normal

In 1921 and 1922, Lewis and Davies [13] reported a study of twenty-two cases of dementia praecox in which the thyroid feeding test was

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11. Weiss, R. F.: Psychoneurotische Störungen bei Hyperthyroidismus, Ztschr. f. klin. Med. **97**:366, 1923.

12. Bowman, K. M., and Fry, C. C.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **14**:819 (Dec.) 1925.

13. Lewis, N. D. C., and Davies, G. R.: Correlative Study of Endocrine Imbalance and Mental Diseases, J. Nerv. & Ment. Dis. **54**:385, 493, 1921; **55**:13, 1922.

performed. Evidence of hyperthyroidism was found in four cases. One of the patients showed definite symptoms of exophthalmic goiter. Thyroidectomy was performed in this case; but a change in the mental condition did not result, although physical symptoms were relieved. Two of the other patients with hyperthyroidism were fed with suprarenal gland; one, with suprarenal and pituitary gland. One of the patients showed marked improvement with regard to mental symptoms and was discharged as recovered. In twelve cases of this series there was definite evidence of hypothyroidism. The thyroid feeding test was found to be of definite value in distinguishing between cases of hyperthyroidism and hypothyroidism.

RESULTS

During the past five years, I have operated on fourteen mentally unbalanced patients with toxic goiter. The mental symptoms were extremely pronounced in the majority of cases, yet all but two patients recovered completely and were able to resume their former occupations. They have remained clear mentally to date.

In two instances, both in young persons, a diagnosis of dementia praecox was made and a poor prognosis given; yet both patients recovered. In all but one case, the derangement occurred in persons suffering from the exophthalmic type of goiter. The exception was a case of the toxic adenomatous type, also with evidence of hyperplasia.

The period during which mental recovery took place varied from several months to a year. Two patients with dementia were not improved by operation; one died in an asylum, the other is still an inmate. In one case, a psychosis developed on the eighth day following removal of an extremely toxic goiter, and this patient is still in a sanitarium.

REPORT OF CASES

CASE 1.—A white woman, aged 35, single, was first seen at the Connecticut State Insane Asylum, Middletown, Conn., when she was in a highly excited state and could not orient herself as to time or place. During the preceding six months, she had lost 40 pounds (18.1 Kg.).

Examination revealed an enlarged thyroid gland, of the consistency found in hyperplastic goiter, with an adenoma about the size of an egg overlying the right lobe. Tremor was pronounced. The pulse rate varied between 140 and 160 and was weak.

The patient was removed to the Good Samaritan Hospital, Cincinnati, being accompanied on the trip by her sister and a nurse. During the journey she made several attempts to leave the train and was continually muttering unintelligibly. While in the hospital at Cincinnati, she tried several times to eat her own feces.

With rest and the administration of a compound solution of iodine she began to improve physically, but her mental condition remained the same. Two weeks after her arrival, thyroidectomy was performed. The reaction was slight and the postoperative course uneventful. Following operation, she was removed to a

sanitarium in one of the suburbs, where she remained for a month. There was great physical improvement during this period; the weight increased and the pulse rate came down to normal.

She was then taken to her sister's home in Cincinnati, and immediately her mental condition began to become clear. Three months after the operation, her mind appeared entirely normal. The following month, she took a motor trip East by herself without suffering any mishap. She has been under observation for a year since that time and is now entirely well. Judging by her present condition, there is every reason to believe that she will remain so.

It would seem that in this case the mental disturbance belonged to the group of exhaustion psychoses. Apparently thyroidectomy, by removing the source of toxemia, was the essential factor in restoring mental balance.

CASE 2.—A white woman, aged 30, married, exhibited a mental disturbance characterized by a marked paranoid trend. For four years she had noticed a fulness in her neck. During the past year this growth had increased in size and she had become emotional and irritable. Since the birth of her last child fourteen months previously, she had lost 20 pounds (9 Kg.)

During the last three months the nervous symptoms had increased perceptibly, and her husband reported that at times her conduct was unusual. On one occasion she left home and was found late at night wandering in a nearby wood. She had loose, poorly organized delusions of persecution, such as are commonly observed in the paranoid type of dementia praecox. She believed that everybody was against her and feared that her husband was trying to kill her with poison. For this reason she refused to take medicine. The family history was negative with regard to mental disease and goiter.

On examination the patient appeared well developed but considerably below normal weight. She looked excited and apprehensive. Her appearance was typical of thyrotoxicosis. Her temperature was 99 F.; her pulse rate, from 130 to 140. The thyroid gland was about three times the normal size and of the consistency of a hyperplastic goiter.

After the usual preparation with a compound solution of iodine, thyroidectomy was performed. After one week she was taken home. Physical improvement was marked, but the mental derangement became worse. After about three months of treatment, however, during which time she ran away from home twice and frequently became so excited that hypnotics were required, she began to improve slowly. This improvement continued over a period of a year, at the end of which her mental status seemed normal. Eighteen months after operation, she was entirely well and her mental condition was clear.

The mental disturbance at the onset of the illness would suggest dementia praecox. In two other cases this diagnosis had actually been made and a poor prognosis given; yet both patients recovered completely after thyroidectomy.

SUMMARY

With the exception of the thyrogenic psychoses occasionally associated with myxedema and cretinism, it is generally held that a direct relationship does not exist between the thyroid gland and mental disease. Careful examination of the thyroid gland in cases of dementia praecox and other psychoses has failed to reveal any consistent anatomic changes.

However, toxic goiter is sometimes observed in association with serious mental diseases, and experience has proved that in such cases, not only the physical condition, but the psychosis as well, may be benefited materially by thyroidectomy. When the mental disturbance is of recent onset and the thyrotoxic manifestations are severe, a fair prospect of mental recovery may be offered following thyroidectomy.

My experience in the treatment for combined toxic goiter and mental alienation comprises fourteen cases, in which I performed thyroidectomy during the past five years. Although the mental symptoms were extremely pronounced in the majority of these cases, all but two of the patients recovered mentally and were able to resume their former occupations, remaining normal to date. In two of the cases, the diagnosis of dementia praecox had been made and a poor prognosis given, yet both patients recovered. In all but one instance, the goiter was of the exophthalmic type; the exception was a toxic adenomatous goiter with evidence of hyperplasia. When thyroidectomy is performed on patients with psychoses, preoperative rest, medication with a compound solution of iodine and the most perfect operative technic are perhaps even more important than in patients with normal mentality.

VITAL CAPACITY

ITS SIGNIFICANCE IN RELATION TO POSTOPERATIVE PULMONARY COMPLICATIONS *

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Determinations of vital capacity, heretofore utilized chiefly by physiologists or restricted to the study of serious cardiac or pulmonary disabilities, are now finding a wider application. The ease with which this accurate information is accumulated has greatly facilitated the dissemination of its practical value.

The determination of the vital capacity of any given patient is clearly one method of appraising the condition present. Any disorder which affects the cardiovascular or respiratory systems reduces the vital capacity. Such determinations may be of use not only in the diagnosis of these cardiorespiratory afflictions, but they also may be of assistance in determining the fitness of any given person. This may be utilized in surgical procedures to appraise the ability of a patient to withstand an operation. Since so many operations of today are those of election, the acquisition of this information seems desirable. Accurate knowledge of pulmonary and cardiac reserve energy might prove to be of inestimable value in deciding on the operability of patients before subjecting them to prolonged procedures. It would seem especially applicable to elderly or debilitated patients and to those whose past history reveals evidence of previous cardiac or pulmonary pathologic conditions. Already articles have appeared relative to the effect of certain surgical conditions and different types of operative incisions on the vital capacity. These latter studies are of great importance since they give a rough estimate of the reduction that follows certain incisions, the position of which interferes with free respiration. Such studies create normal base lines below which further reductions have a deeper significance.

The startling increase in the number of postoperative pulmonary complications during the past two decades points to the importance of making accurate observations during the convalescent period. Pfannenstiel,[1] in 1903, and Otte,[2] in 1907, reported large series of cases in which postoperative pulmonary complications were not observed, a

* From the Surgical Clinic of the Lakeside Hospital, Cleveland.

1. Pfannenstiel, J.: Ueber die Vorzüge der Äthernarkose, Zentralbl. f. Gynäk. **1**:8, 1903.

2. Otte, A.: Ueber die postoperativen Lungenkomplikationen und Thrombosen nach Aethernarkosen, München. med. Wchnschr. **54**:2473, 1907.

remarkable and praiseworthy record. In the light of present knowledge, however, it is possible that a reexamination of the individual case reports might reveal minor symptoms indicative of transient pulmonary complications.

In 1906, Armstrong [3] reported a pulmonary morbidity of 2.12 per cent in 2,500 cases at the Montreal General Hospital. In 1908, von Lichtenberg [4] collected 23,673 cases with a pulmonary morbidity of 1.9 per cent. The same year, Ranzi [5] published 263 pulmonary complications from 6,871 operations in von Eiselberg's Clinic at Vienna, a morbidity of 3.8 per cent. Beckman [6] reported the pulmonary complications at the Mayo Clinic in 1910, 1912 and 1913. When his figures are amended to include pulmonary embolism, they reveal morbidities of 1.12 per cent, 1.57 per cent and 1.27 per cent, respectively. Cutler and Morton [7] found 1.86 per cent of cases in 3,490 operations at the Massachusetts General Hospital in 1917. One year later, McKesson [8] computed 3.03 per cent of pulmonary complications from the combined statistics of 39,438 operations. In 1920, Cutler and Hunt [9] reported a pulmonary morbidity of 3.52 per cent in 1,562 patients operated on at the Peter Bent Brigham Hospital, and the following year,[10] 3.92 per cent in 1,604 cases at the same hospital.

It seems probable that a more careful analysis of all these statistics would reveal not actually a greater number of postoperative complications during recent years, but a more astute recognition and correct interpretation of symptoms which heretofore have passed unnoticed. A slight unexplained fever, a transient cough with or without thoracic pain, a few flecks of blood-tinged sputum without gross clinical evi-

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3. Armstrong, G. E.: Lung Complications After Operations with Anesthesia, Brit. M. J. **1**:1141, 1906.

4. Von Lichtenberg, A.: Die postoperativen Lungenkomplikationen, Centralbl. f. d. Grenzgeb. d. Med. u. Chir. **11**:129, 1908.

5. Ranzi, E.: Ueber postoperative Lungencomplicationen embolischer Natur, Arch. f. klin. Chir. **87**:380, 1908.

6. Beckman, E. H.: Pulmonary and Circulatory Complications Following Surgical Operations, Collected Papers Mayo Clinic, Philadelphia, W. B. Saunders Company, 1910, pp. 594-599; Complications Following Surgical Operations, ibid., 1912, pp. 738-745; Complications Following Surgical Operations, ibid., 1913, pp. 776-788.

7. Cutler, E. C., and Morton, J. J.: Postoperative Pulmonary Complications, Surg. Gynec. Obst. **25**:621, 1917.

8. McKesson, E. I.: Some Observations on Postoperative Lung Complications, Am. J. S. (Anes. Supp.) **32**:16, 1918.

9. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Surg. **1**:114 (July) 1920.

10. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Int. Med. **29**:449 (April) 1922.

dence of a pathologic process in the chest could well have escaped recognition.

Even such an extensive lesion as massive atelectasis of one lung passed unrecognized for many years by physicians and surgeons alike. The condition was first described by Pasteur [11] in 1908, but the first cases of postoperative collapse to be reported in the American literature did not appear until 1921.[12]

These studies on the vital capacity of surgical patients were undertaken with the hope of establishing a means of detecting transient, minor postoperative upsets and corroborating the diagnosis of gross pulmonary complications.

METHOD

Dr. Francis Peabody [13] stated:

"The vital capacity of the lungs consists of the volume of the deepest possible expiration after the greatest possible inspiration. A certain amount of air, known as the residual air, remains in the lungs after complete expiration but the vital capacity represents the degree of movability of the lungs and anything which interferes with the movement of the lungs, preventing the entrance of the air into them, causes a decrease in the vital capacity."

Any acute process in the abdomen producing reflex spasm of the abdominal musculature involves the accessory muscles of respiration and hence inhibits respiratory excursion. Likewise, any operative procedure attended subsequently by splinting of the abdomen has a direct influence on the vital capacity. Consequently, it was necessary first to establish normal curves in order to determine the reduction in vital capacity following the various types of operative procedures in uncomplicated cases.

All the male patients admitted to the surgical service of the Lakeside Hospital were studied during a period of several months. At first the female patients were included, but the men were found to be more cooperative and the results accordingly more satisfactory. All the patients were encouraged to exert themselves to the utmost in spite of pain, and a friendly spirit of rivalry was stimulated in the ward, each man trying to "blow harder" than his neighbor in the next bed. Whenever it was felt that a patient was not cooperating fully, his observations were discarded.

The Collins Vital Capacity Spirometer was used. It consists of a delicately compensated bell floating in a cylinder of water. The most essential feature of the apparatus is the direct reading dial which is graduated to tenths of liters. Fractions of these graduations may be estimated in tenths also. As the bell rises with expiration, a movable pointer passes over the face of the dial, stops at the highest point and remains in that position. A second pointer is connected with the dial which is of value if the patient tends to inspire from the spirometer just before expiring into it. In such a case, the bell may be raised and the pointers adjusted so that both are at the 1 liter mark. If the

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11. Pasteur, W.: Massive Collapse of the Lung, Lancet **2**:1351, 1908.

12. Scrimger, F. A. C.: Postoperative Massive Collapse of the Lung, Surg. Gynec. Obst. **32**:486, 1921.

13. Peabody, F. W.: The Clinical Importance of the Vital Capacity of the Lungs, Northwest Med. **22**:307, 1923.

patient inspires from the apparatus before expiring, one pointer moves toward the zero mark until inspiration ceases. The other moves in a clockwise direction with expiration. In this case, the difference between the two pointers represents the vital capacity of the lungs. The calibration of the apparatus is exact and the breathing tube is sufficiently large to prevent resistance to expiration.

A nose clip was not used; the patient was instructed to close his nose by gentle pressure of the thumb and finger. Whenever leakage occurred, it could be easily detected by fogging on the polished metal surface of the mouthpiece.

All observations were made with the patients in the recumbent position and are accordingly slightly lower than the average recorded for patients of equal height and weight in the erect posture.[14] They were also taken at least two hours after a meal in order to obviate the possible inhibitory effect of abdominal distention.

The vital capacity was taken on admission and daily throughout the postoperative convalescent period, until a high level was reached or the patient discharged.

The highest of three to five observations was chosen as the most accurate. A record was kept of the operative procedure, the type and the duration of the anesthethic. There were no untoward wound results, even in patients with several drains in the abdomen. Clean wounds healed firmly by primary union as usual. A careful clinical record was kept throughout the patient's sojourn in the hospital and correlated with the vital capacity each day. As an index of the patient's general condition the temperature curve has been plotted beneath the vital capacity.

OBSERVATIONS ON NORMAL PATIENTS

All the similar types of cases have been grouped together. The preoperative level in each has been arbitrarily regarded as 100 per cent and the daily reductions calculated in percentages of this figure.

Chart 1 represents the composite curve of eight uncomplicated operations for bilateral inguinal hernia. Table 1 gives the daily readings for each patient in detail with the average and percentage calculated below.

Chart 2 is a record of the vital capacity and temperature in eight uncomplicated operations for unilateral inguinal hernia. Table 2 is a detailed representation of the daily readings for each patient with the average and percentage calculated below.

Chart 3 and table 3 are representative of eight patients operated on through a McBurney incision for acute appendicitis. The higher readings during the latter days of the convalescence indicate a diminished vital capacity on admission. This was undoubtedly caused by the limitation of respiratory excursion incidental to an acute process in the abdomen.

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14. Christie, C. D., and Beams, A. J.: The Estimation of Normal Vital Capacity, Arch. Int. Med. **30**:34 (July) 1922.

Chart 4 represents the average vital capacity and temperature of five patients who were subjected to upper abdominal operations. Table 4 presents the daily readings in detail. The third patient in the series had a perforated gastric ulcer. On admission, there was marked

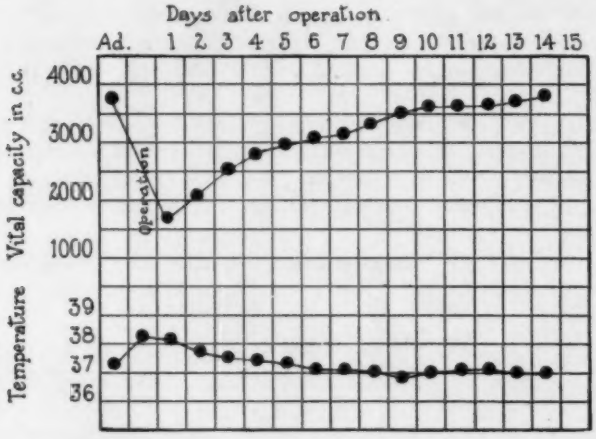

Chart 1.—Composite curve showing the vital capacity of eight patients who were operated on for bilateral inguinal hernia.

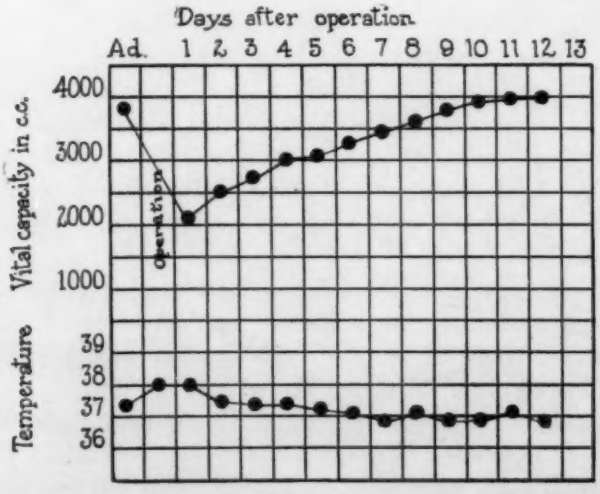

Chart 2.—Curve showing the vital capacity and temperature in eight uncomplicated cases in which operation for unilateral inguinal hernia was performed.

spasm of the whole abdomen and likewise a striking decrease in the vital capacity, 1,800 cc. compared with 5,420 cc. on the fifteenth postoperative day.

There was only one uncomplicated operation involving a midline incision in the lower part of the abdomen. Resection of the bladder and transplantation of the ureters into the rectum were performed for carcinoma of the bladder. This curve is presented in chart 5.

Seven patients were operated on for hydrocele. A slight reduction in the vital capacity occurred during the first few days after operation, in spite of the fact that the aponeurosis of the external oblique was not

TABLE 1.—*Vital Capacity of Patients Operated On for Bilateral Inguinal Hernia Given in Cubic Centimeters*

On Admission	Days After Operation													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
3,560	2,250	2,690	2,950	2,990	3,050	3,230	3,150	3,000	3,250	3,300	3,560	3,700	3,660	3,700
3,300	1,720	2,200	2,580	2,310	2,630	2,720	2,900	3,020	3,150	3,100	3,400	3,500	3,410	3,520
5,000	2,440	2,700	3,800	4,230	4,320	4,450	3,900	4,450	4,800	5,090	4,150	3,900	4,500	4,850
2,350	1,900	1,980	2,100	2,500	2,530	2,600	2,730	2,740	2,720	2,800	2,810	2,920	2,930	2,930
3,500	1,000	1,000	1,320	1,740	1,800	2,120	2,680	2,990	3,000	3,180	3,400	3,500	3,500	3,500
3,900	1,000	1,790	1,810	2,600	2,720	2,700	2,800	3,100	3,500	3,750	3,550	3,600	3,600	3,910
3,880	1,700	1,820	2,140	2,310	2,510	2,790	3,120	3,200	3,350	3,320	3,380	3,300	3,510	3,610
4,600	1,550	2,950	3,500	3,760	3,810	4,010	4,060	4,300	4,100	4,430	4,600	4,600	4,760	4,750
3,761	1,695	2,141	2,525	2,809	2,921	3,077	3,167	3,350	3,506	3,629	3,696	3,627	3,734	3,846
100%	45%	57%	67%	74%	77%	81%	84%	89%	93%	96%	99%	96%	99%	102%

TABLE 2.—*Unilateral Inguinal Hernia*

On Admission	Days After Operation											
	1	2	3	4	5	6	7	8	9	10	11	12
3,800	1,740	1,980	2,210	2,810	3,190	3,360	3,420	3,680	3,900	3,850	3,900	3,910
3,900	3,570	3,400	3,310	3,410	3,610	3,900	3,930	4,110	4,190	4,270	4,300	4,300
3,600	2,050	2,300	2,710	3,070	3,220	3,520	3,560	3,600	3,600	3,710	3,770	3,820
3,550	1,800	2,740	2,990	3,240	3,140	3,450	3,570	3,670	3,700	3,750	3,760	3,760
3,000	1,700	2,150	2,560	2,640	2,710	2,900	3,000	2,960	3,200	3,250	3,300	3,690
5,300	2,840	3,320	3,320	3,710	3,700	4,060	4,300	4,700	4,900	5,220	5,200	5,190
3,800	2,500	2,800	3,300	3,350	2,820	3,320	3,400	3,480	3,690	3,800	3,870	3,890
3,980	1,840	2,040	2,400	2,400	2,550	2,450	3,000	3,200	3,620	3,880	3,820	3,900
3,250	1,630	1,820	2,100	2,380	2,600	2,910	3,100	3,120	3,320	3,300	3,400	3,400
3,806	2,188	2,505	2,766	3,000	3,090	3,319	3,489	3,630	3,791	3,902	3,924	3,994
100%	57%	65%	72%	78%	81%	87%	91%	95%	99%	102%	103%	104%

incised. Curves are represented in chart 6 and daily readings are given in detail in table 5.

Rectal operations, including hemorrhoidectomies, excision of fistulas, incision and drainage of ischiorectal abscesses, do not produce any postoperative reduction in vital capacity. Chart 7 is a composite curve of the vital capacity and temperature of eight patients. Table 6 presents the daily observations in detail.

Operations on the extremities likewise do not have any appreciable effect on the vital capacity other than that which can be explained on the basis of daily variation; (chart 8, table 7). Fifteen patients were studied. The operations include excision of varicose veins, amputations

of fingers and toes, incision and drainage of osteomyelitis, incision, drainage and arthrodesis of joints.

Some interesting results were obtained in patients subjected to thyroidectomy. Chart 9 shows an immediate and striking reduction in

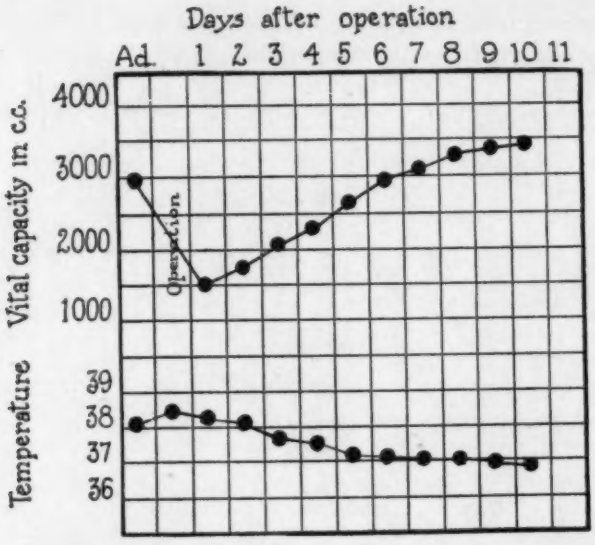

Chart 3.—Vital capacity of eight patients on whom appendicectomy was performed (McBurney).

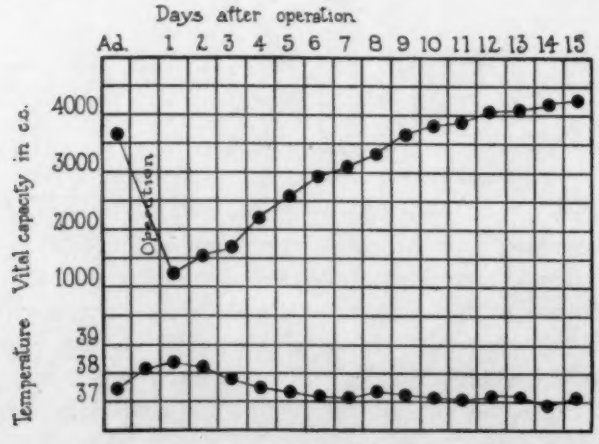

Chart 4.—Average vital capacity and temperature of five patients who were subjected to operation on the upper part of the abdomen.

vital capacity followed by a rapid recovery. Table 8 is a detailed analysis of each case.

Other operations on the neck have only a slight effect on the vital capacity. This lies within the limits of normal daily variation and

probably has no bearing on the operative procedure. The four cases include excision of tuberculous glands, incision and drainage of an abscess, and radical dissection (chart 10, table 9).

OBSERVATIONS ON PATIENTS WITH POSTOPERATIVE COMPLICATIONS

There were no complications among the patients operated on for bilateral inguinal hernia.

Chart 11 is a graph of the vital capacity and temperature of a healthy man, aged 26, who was operated on for right inguinal hernia under gas-oxygen-ether anesthesia. The usual reduction in vital

TABLE 3.—*Acute Appendicitis (McBurney Incision)*

On Admission	Days After Operation									
	1	2	3	4	5	6	7	8	9	10
2,540	1,420	1,350	1,850	1,910	2,100	2,500	2,600	2,820	2,820	2,800
1,820	1,410	1,460	1,590	1,720	1,900	2,100	2,400	2,800	2,910	2,910
3,120	1,360	1,400	1,580	1,700	2,100	2,350	3,000	2,850	3,160	3,150
4,050	1,550	1,840	2,310	2,600	3,080	3,460	3,610	3,820	4,060	4,260
3,480	1,680	1,940	2,000	2,850	3,430	3,610	3,760	4,030	4,100	4,100
3,600	2,060	2,580	2,970	3,310	3,510	3,680	3,980	3,960	3,950	3,960
2,110	1,710	1,900	1,980	2,040	2,300	2,340	2,600	2,450	2,600	2,600
3,100	1,080	2,080	2,490	2,910	3,150	3,510	3,510	3,710	3,700	3,710
2,977	1,534	1,819	2,108	2,380	2,696	2,944	3,184	3,304	3,413	3,436
100%	52%	61%	70%	79%	90%	98%	106%	110%	114%	115%

TABLE 4.—*Operations on the Upper Part of the Abdomen*

On Admission	Days After Operation														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
3,680	900	1,300	1,100	1,760	2,300	3,000	2,850	3,390	3,750	3,650	3,700	3,750	3,750	3,750	3,750
3,830	1,690	1,950	2,450	2,760	2,720	2,880	3,000	3,260	3,500	3,450	3,500	3,760	3,520	3,900	3,900
1,800	1,350	2,460	2,840	3,410	3,720	4,450	5,130	4,950	5,210	5,370	5,380	5,420	5,420	5,410	5,420
3,380	1,060	980	1,300	1,640	1,760	2,080	2,200	2,500	2,700	2,950	2,980	3,050	3,210	3,320	3,300
5,430	1,250	1,060	950	1,760	2,150	2,230	2,720	2,820	3,160	3,590	3,820	4,350	4,520	4,730	5,100
3,624	1,248	1,550	1,724	2,266	2,530	2,924	3,180	3,384	3,682	3,802	3,876	4,066	4,084	4,222	4,294
100%	34%	43%	48%	63%	70%	81%	88%	93%	101%	105%	107%	112%	112%	116%	118%

capacity occurred on the first postoperative day. When the observations were taken on the forenoon of the second day a marked secondary reduction was found. During the early afternoon, the patient developed acute respiratory distress; temperature, pulse and respirations were elevated, and physical examination of the chest revealed a massive atelectasis on the right side. This was confirmed by a roentgenogram. On the third day, a slight increase in vital capacity and fall in temperature were observed. On the fourth day, the vital capacity was again reduced and the temperature elevated. Following this, both rapidly returned to normal (compare with chart 2).

Chart 12 is most interesting. The patient was a well developed, but somewhat plethoric man, aged 44, who had suffered with chronic asthma

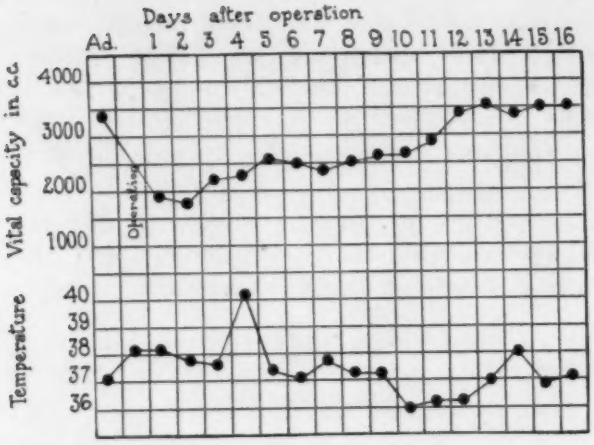

Chart 5.—Vital capacity of one patient following resection of the bladder and transplantation of the ureters.

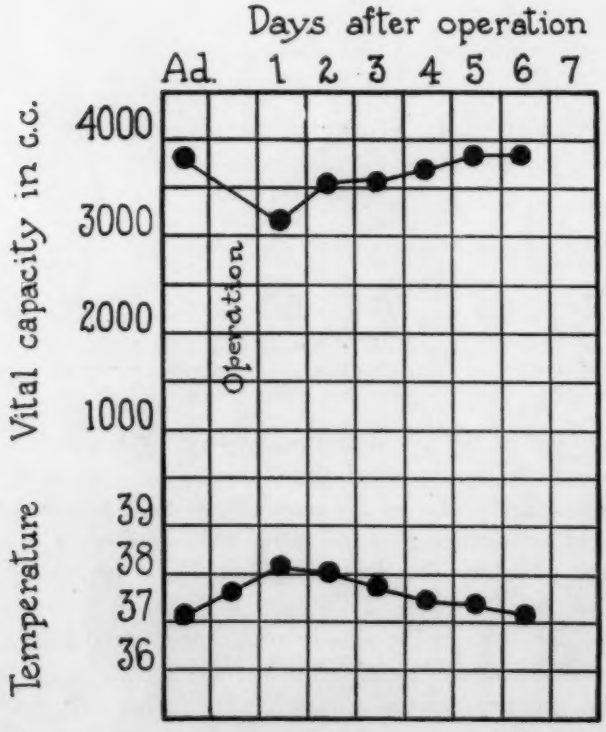

Chart 6.—Vital capacity of seven patients operated on for hydrocele.

for many years. Examination of the chest revealed typical asthmatic symptoms; clubbing of the fingers and toes was present. The preoperative vital capacity was only 2,650 cc., 64 per cent of the expected normal for a man of corresponding height and weight. A right inguinal hernia was repaired under local infiltration and block anesthesia with procaine hydrochloride. The usual reduction in vital capacity occurred on the first postoperative day, accompanied by a marked elevation in temperature. During the subsequent four days, the vital capacity increased slowly, but the temperature remained elevated. On the sixth day, a decrease in vital capacity was noted. During the next three

TABLE 5.—*Hydrocele*

On Admission	Days After Operation					
	1	2	3	4	5	6
4,660	4,100	4,160	4,200	4,680	4,680	4,660
3,610	3,170	3,200	3,140	3,300	3,350	3,550
3,800	2,500	2,700	2,950	3,200	3,510	3,810
3,470	3,140	3,500	3,550	3,550	3,550	3,550
4,500	4,200	4,700	4,520	4,600	4,600	4,660
3,560	2,200	3,280	3,310	3,520	3,620	3,400
2,980	3,140	3,120	3,340	3,320	3,340	3,340
3,797	3,207	3,536	3,573	3,739	3,807	3,853
100%	84%	98%	94%	98%	100%	101%

TABLE 6.—*Rectal Operations*

On Admission	Days After Operation				
	1	2	3	4	5
4,200	4,080	4,500	4,530	4,550	4,500
3,170	3,500	4,170	4,200	4,210	4,350
3,680	3,600	3,680	3,670	3,690	3,680
4,210	4,120	4,250	4,100	4,100	4,170
4,480	4,680	5,000	4,800	4,900	4,950
4,300	4,320	4,320	4,500	4,440	4,440
4,480	4,100	4,560	4,510	4,560	4,560
3,320	3,120	3,450	3,420	3,450	3,450
4,116	3,940	4,252	4,216	4,238	4,274
100%	99%	103%	102%	103%	104%

days, the vital capacity continued steadily downward, the temperature rose slightly, and the patient showed clinical signs of bronchopneumonia. This diagnosis was confirmed by the roentgen-ray examination. Recovery was extremely slow. The temperature remained elevated until the twenty-seventh postoperative day and the vital capacity did not attain its preoperative level until the twenty-ninth day.

In spite of this stormy convalescence, the patient wished to have a left femoral hernia repaired before leaving the hospital. After the temperature had been normal for several days, this operation was performed under local and block anesthesia as before. The usual reduction in vital capacity occurred on the first postoperative day and a marked

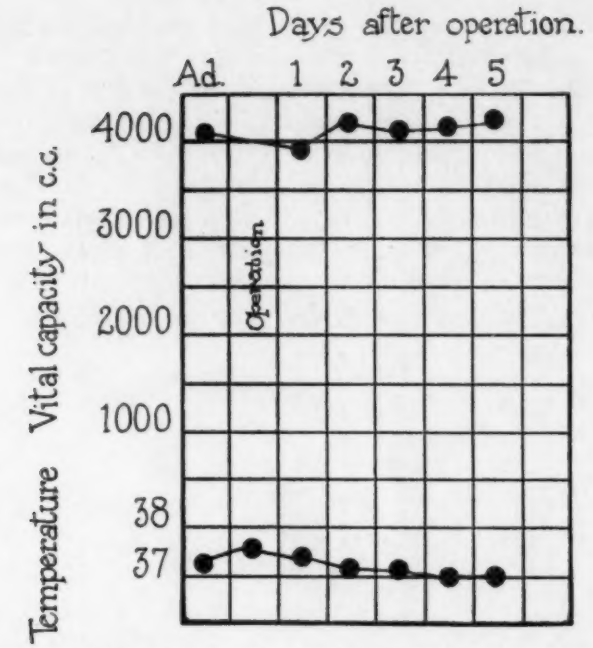

Chart 7.—Composite curve of the vital capacity and temperature of eight patients on whom rectal operations were performed.

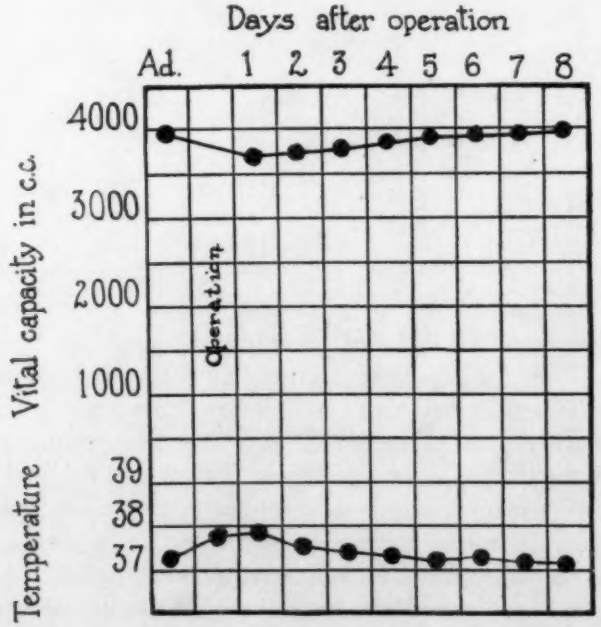

Chart 8.—Vital capacity of fifteen patients who were subjected to operations on the extremities.

rise in temperature similar to that observed after the first operation. The temperature rapidly subsided, however, and the physical examination did not reveal evidence of any pulmonary complication. The vital capacity showed a normal upward trend and recovered its preoperative level on the tenth day.

Chart 13 represents the vital capacity and temperature of a youth, aged 21, operated on for acute appendicitis. There was a marked fall in the vital capacity on the first postoperative day and a rapid recovery

TABLE 7.—*Operations on the Extremities*

On Admission	Days After Operation							
	1	2	3	4	5	6	7	8
4,400	4,460	4,400	4,400	4,500	4,480	4,480	4,480	4,480
3,220	3,150	3,240	3,320	3,370	3,320	3,360	3,410	3,380
4,100	4,350	4,340	4,350	4,350	4,350	4,350	4,350	4,350
3,290	2,050	2,140	1,900	2,300	2,600	2,400	2,750	2,890
4,800	4,780	4,860	4,860	4,860	4,860	4,860	4,860	4,860
3,490	3,450	3,460	3,400	3,510	3,450	3,510	3,510	3,510
4,210	4,320	4,290	4,280	4,320	4,320	4,320	4,320	4,320
3,220	2,420	2,510	2,650	3,150	3,290	3,160	3,290	3,260
4,000	3,650	3,750	3,810	3,850	3,700	3,790	3,810	3,850
4,500	4,610	4,720	4,710	4,710	4,710	4,710	4,710	4,710
3,560	3,300	3,520	3,600	3,700	3,760	3,800	3,880	3,790
4,050	3,630	3,180	3,620	3,800	3,840	3,930	3,900	4,000
4,900	4,600	4,850	4,880	4,900	4,900	4,900	4,900	4,900
2,610	2,040	2,520	2,390	2,600	2,600	2,450	2,550	2,670
4,710	4,430	4,420	4,890	4,750	4,790	5,000	5,000	5,000
3,937	3,716	3,747	3,804	3,911	3,931	3,935	3,981	3,998
100%	94%	95%	96%	99%	100%	100%	101%	101%

TABLE 8.—*Thyroidectomy*

On Admission	Days After Operation							
	1	2	3	4	5	6	7	8
4,790	2,100	3,400	3,860	4,140	4,300	4,520	4,800	4,900
3,200	1,360	1,660	2,350	2,800	2,900	3,120	3,220	3,220
4,500	1,800	2,090	3,100	3,560	4,150	4,500	4,500	4,500
4,400	910	1,750	2,840	3,000	4,420	4,400	4,400	4,400
4,350	3,450	3,100	3,640	4,110	4,220	4,360	4,300	4,350
4,248	1,924	2,400	3,158	3,642	3,998	4,180	4,280	4,274
100%	45%	57%	74%	86%	94%	98%	101%	101%

which on the eighth and ninth days had not reached the preoperative level. The temperature remained elevated and on the ninth day rose still higher to 39.5 C. (103 F.). On the tenth day, a marked reduction in vital capacity occurred but no clinical evidence of a pathologic process in the chest could be found. The temperature continued upward; on the eleventh day, there was a further reduction in vital capacity and physical examination revealed bronchopneumonia. In two days, the vital capacity fell from 3,920 cc. to 610 cc. During the next twelve days, the temperature gradually subsided to normal and the vital capacity increased to 2,690 cc. On the twenty-third day, the patient complained of thoracic pain on deep inspiration; the vital capacity fell to 1,290 cc.

and the temperature rose to 39 C. (102.1 F.). The physical examination was negative. The following day, the temperature was 40.6 C. (105 F.) and the vital capacity 820 cc. The physical examination and roentgenogram revealed pleurisy with effusion at the base of the right lung. Thoracentesis was performed twice during the next five days. Following this, the temperature fell by lysis, and the vital capacity gradually increased but did not attain its preoperative level. The patient was discharged on the forty-third postoperative day. On admission, the vital capacity was 4,450 cc.; on discharge 3,000 cc. (compare with chart 3).

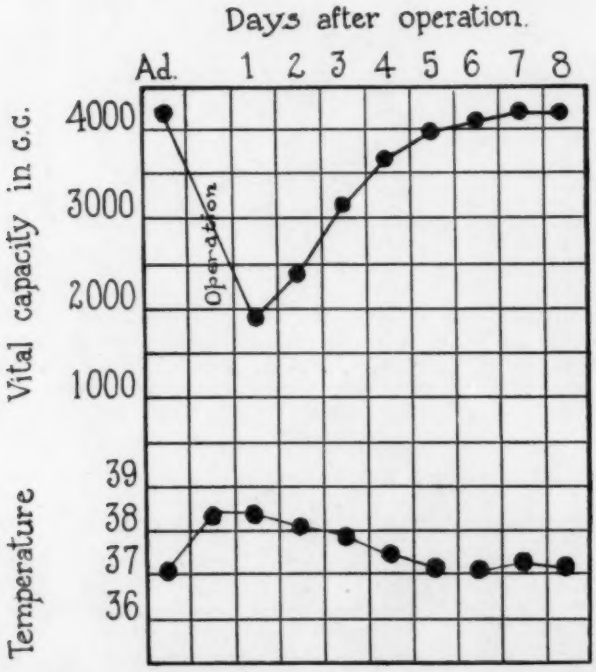

Chart 9.—Vital capacity of five patients on whom thyroidectomy was performed.

Chart 14 indicates the effect of postoperative abdominal complications on vital capacity. The patient was a well developed and well nourished man, aged 49. Following appendicectomy and drainage of an abscess under gas-oxygen-ether anesthesia, the vital capacity remained depressed and the temperature elevated. From this low level, a definite downward trend was noted on the sixth day, and rectal examination revealed a pelvic abscess. This was drained through the abdomen the following day. The vital capacity continued its downward course, and two days after the second operation, general peritonitis was present.

During the next five days, the vital capacity remained at essentially the same level and the temperature at 38 C. (100.4 F.). The patient was extremely sick; his pulse was weak and rapid; the abdomen was spastic and distended. On the sixth day after the development of general peritonitis, a definite improvement occurred in his clinical condition; the vital capacity was higher and the temperature lower. From then onward there was a gradual but steady upward trend to the vital capacity curve. The temperature returned to normal almost immediately (compare with chart 3).

Chart 15 illustrates the effect on the vital capacity of appendicectomy complicated by general peritonitis. The patient was a well developed and well nourished colored man, aged 26. The preoperative vital capacity was somewhat below normal. Because of this, the subsequent decrease on the first postoperaive day was less than usual. On the second day, the patient showed clinical evidence of generalized peritonitis and a further reduction in vital capacity occurred. During the next

TABLE 9.—*Other Operations on the Neck*

On Admission	Days After Operation					
	1	2	3	4	5	6
3,610	3,400	3,850	3,820	3,900	4,000	4,020
3,620	3,440	3,510	3,690	3,720	3,720	3,720
3,380	3,300	3,420	3,700	3,640	3,700	3,700
3,720	3,620	3,750	3,670	3,620	3,800	3,840
3,582	3,440	3,632	3,720	3,720	3,805	3,820
100%	96%	101%	104%	104%	106%	106%

three days, his vital capacity and clinical condition remained essentially the same. On the sixth postoperative day, a slight improvement was noted in both. Recovery was slow. He was discharged on the twenty-first day; the vital capacity was 3,200 cc. on discharge, 1,850 cc. on admission (compare with chart 3 and 14).

Chart 16 represents the vital capacity and temperature of a white man, aged 34, subjected to cystectomy and transplantation of the ureters into the rectum because of carcinoma of the bladder. The usual reduction in vital capacity occurred on the first postoperative day, accompanied by a rather marked elevation of temperature. On the third day, the vital capacity had increased slightly and the temperature had subsided to 38.2 C. (100.7 F.). On the fourth day, however, a secondary reduction in vital capacity was apparent and the temperature had again risen to 38.8 C. (101.4 F.). Examination of the patient revealed evidence of general peritonitis. The following day, there was a slight improvement in the vital capacity. This was only temporary, however, and the general trend was downward until death occurred on the thirteenth day after operation (compare with chart 5).

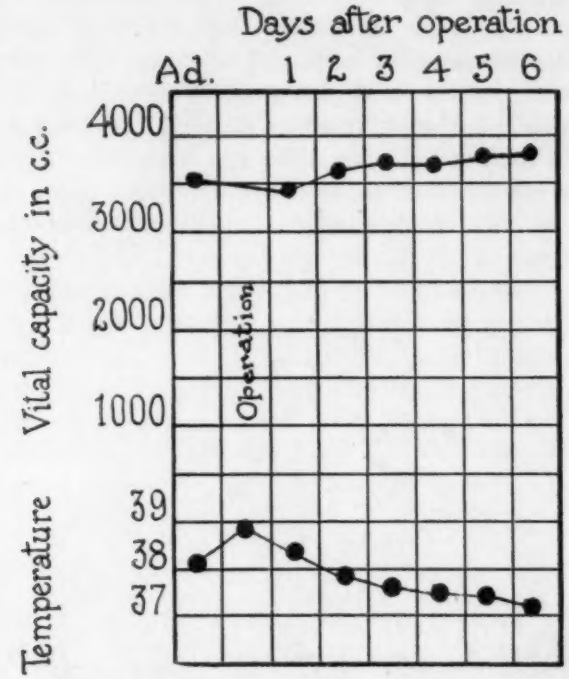

Chart 10.—Vital capacity of four patients subjected to other operations on the neck.

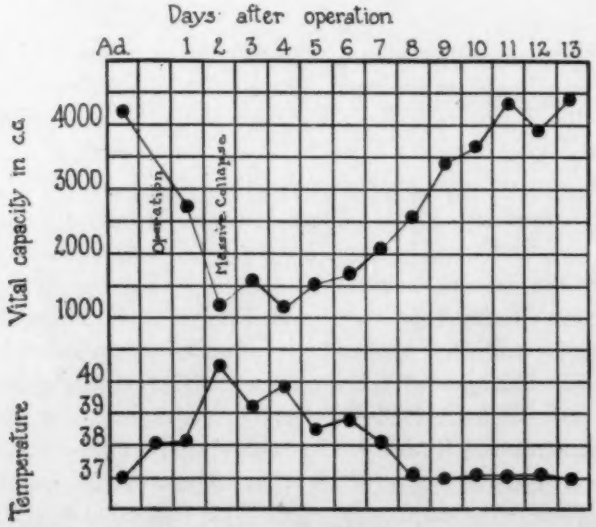

Chart 11.—Vital capacity and temperature of a patient on whom operation for unilateral hernia was followed by massive collapse of the right lung.

No other postoperative complications developed, and consequently no curves are available for comparison with normal curves following operations on the upper part of the abdomen, extremities, neck or rectum.

COMMENT

Since this work was started, two articles have appeared on vital capacity before and after operation. The curves presented in this paper compare favorably with those of Churchill and McNeil [15] and Head.[16]

It is evident from an analysis of the charts and tables that abdominal operations produce a tremendous immediate decrease in vital capacity followed by a gradual return to normal. There is an apparent similarity in the curves for each type of operative incision.

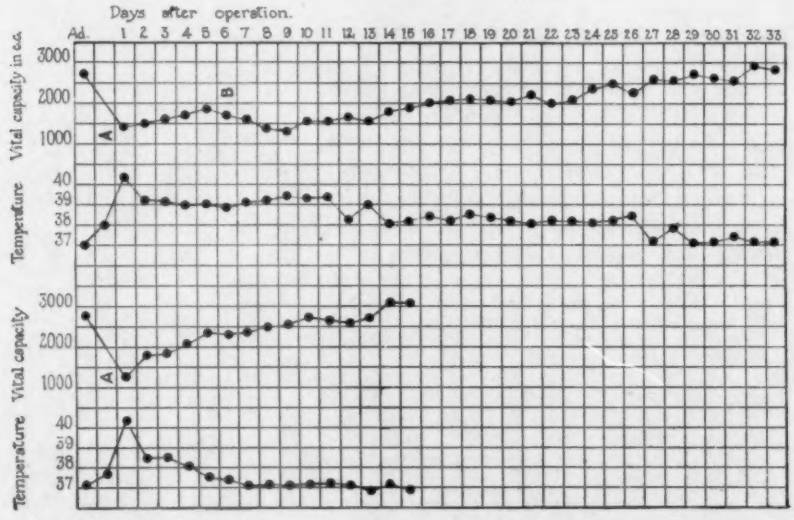

Chart 12.—Vital capacity and temperature curves of a patient operated on for right inguinal hernia; postoperative bronchopneumonia followed; an operation for left femoral hernia was attended by a normal convalescence. *A* indicated operation; *B*, pneumonia.

Partial removal of the thyroid gland likewise entails an immediate and pronounced fall in the vital capacity followed by a rapid recovery. All other operations on the neck and those on the extremities and rectum have no effect on the vital capacity. The reason is obvious; the muscles of respiration are not involved in the operative incision. Why the vital capacity should be so profoundly, though temporarily, affected by

15. Churchill, E. D., and McNeil, D.: The Reduction in Vital Capacity Following Operation, Surg. Gynec. Obst. **44**:483, 1927.

16. Head, J. R.: The Effect of Operation Upon the Vital Capacity, Boston M. & S. J. **197**:83, 1927.

thyroidectomy is hard to explain. Possibly exposure of the trachea, retention of mucus, congestion and edema of the air passages may have some bearing on it. It may be that the excitement, apprehension and psychic instability characteristic of the majority of patients after thyroidectomy secondarily restrict their voluntary respiratory control.

By far the greatest reduction occurs when the operative incision is located in the upper part of the abdomen and invades the epigastric triangle. There is still a veil of mystery obscuring the accuracy of knowledge regarding the effect of upper abdominal operations. Cutler

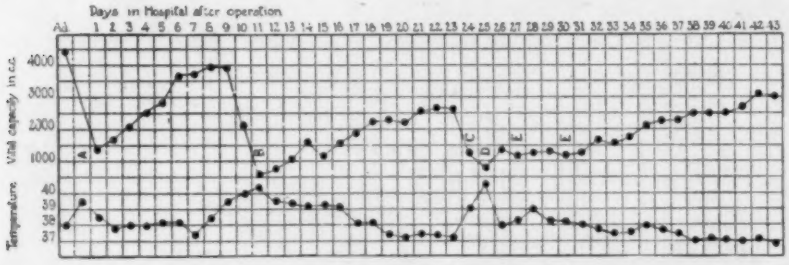

Chart 13.—Vital capacity and temperature curves of a patient on whom appendicectomy was performed; bronchopneumonia and pleurisy with effusion followed. *A* indicates operation; *B*, bronchopneumonia; *C*, pain on effusion; *D*, pleurisy with effusion; *E*, thoracentesis.

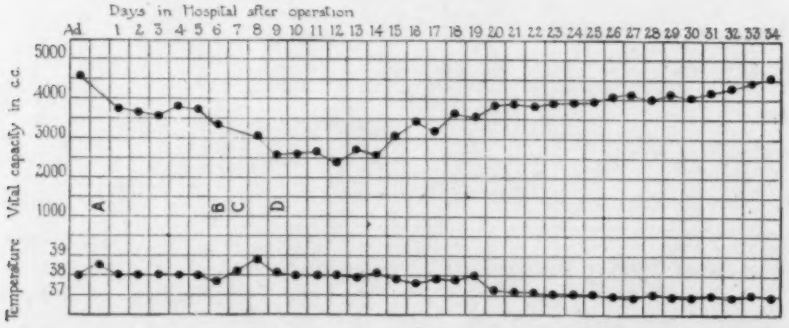

Chart 14.—Vital capacity and temperature curves of a patient on whom appendicectomy was performed; a residual pelvic abscess was drained and general peritonitis developed. *A* indicated operation; *B*, pelvic abscess; *C*, drainage and *D*, general peritonitis.

and Hunt [17] found that postoperative pulmonary complications occur in 8 per cent of epigastric operations, 4 per cent of laparotomies and 2 per cent of all operations. They attributed this to greater mobility of the field during operation and the consequent greater freedom with which multiple small emboli may be dislodged. Subsequent to epigastric

17. Cutler and Hunt (footnote 9 and 10).

operations, there is a more complete and prolonged splinting of the abdomen and lower part of the thorax, with the result that the respiratory movements are more grossly impaired over a longer period of time than after operations on any other part of the body. May not this pulmonary embarrassment and comparative immobility play some part in the more frequent occurrence of postoperative complications follow-

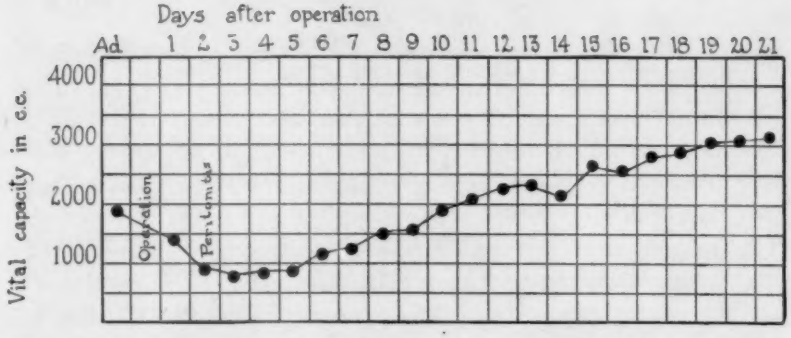

Chart 15.—Vital capacity of a patient subjected to appendicectomy for ruptured appendicitis and general peritonitis.

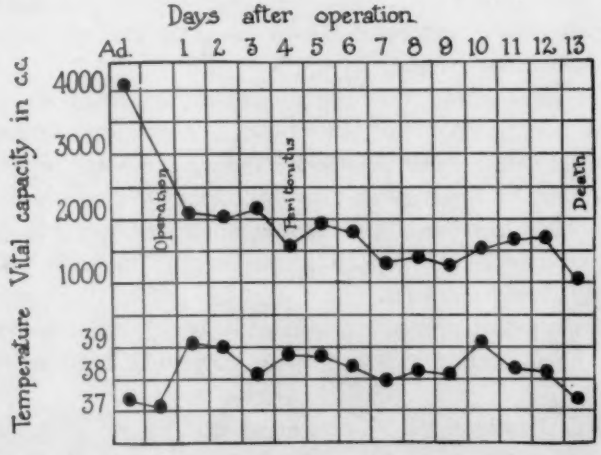

Chart 16.—Vital capacity of a patient subjected to cystectomy and transplantation of the ureters; general peritonitis developed and death ensued.

ing upper abdominal incisions? Karsner and Ash [18] showed experimentally that a partially atelectatic lung offers a more fertile soil for the reception of emboli than one which is normally aerated. The sequence of events seems logical. Most upper abdominal incisions are placed to the right of the midline; consequently, expansion of the lower lobe of

18. Karsner, H. T., and Ash, J. E.: Studies in Infarction: II. Experimental Bland Infarction of the Lung, J. M. Research **22**:205, 1912-1913.

the right lung is most seriously impaired by the subsequent splinting of the upper abdominal musculature. Emboli showered off from a mobile field find a congenial environment in this partially collapsed lobe—bronchopneumonia, pulmonary infarction and abscess of the lung follow, depending on the number and character of the emboli. The old aspiration theory—that complications occur most frequently in the lower lobe of the right lung because of the more vertical position of its major bronchus—no longer seems entirely tenable.

In this series, the patient who developed a massive atelectasis following hernial repair showed a marked secondary reduction in vital capacity before clinical symptoms of the condition were manifest (chart 11). As the lung expanded, recovery was rapid and return to the normal preoperative level required no longer than in the uncomplicated operations for hernia.

The patient who had postoperative pneumonia, showed a slight gradual recovery during the first few days, followed by a secondary fall and slow recovery (chart 12). The patient who developed a late postoperative pneumonia and pleurisy with effusion showed a marked secondary fall in vital capacity, partial recovery as the pneumonic process became organized, and still a third reduction as the pleural cavity became filled with fluid (chart 13). In all these cases the diagnosis of postoperative pulmonary complication was made by the spirometer before clinical signs became evident.

Complications within the abdomen likewise manifest themselves through the medium of the spirometer. Abdominal distention and muscular spasm in general peritonitis reduce the vital capacity. This is partially due to mechanical encroachment of distended abdominal viscera and partly to reflex muscle spasm which restricts deep respiratory excursions (charts 14, 15 and 16). As the inflammation subsides, the vital capacity slowly recovers.

All patients with pathologic processes in which intrathoracic surgical procedures have been performed have purposely been omitted from this study.

The administration of a general anesthetic has no appreciable effect on the vital capacity. Patients operated on under gas-oxygen or a mixture of gas-oxygen and ether showed no greater reduction in vital capacity than those operated on under local anesthesia. Chloroform and ethylene were not used.

CONCLUSIONS

1. Preoperative determinations of vital capacity are suggested as an adjunct in determining the operability of patients.

2. Operations on the extremities and rectum do not have any effect on the vital capacity.

3. Thyroidectomy produces a striking decrease followed by a rapid recovery. Other operations on the neck do not have any effect.

4. A marked reduction in vital capacity occurs after abdominal operations. When plotted, the curves for each type of incision simulate one another.

5. The greatest reduction and most prolonged period of recovery follow operations on the upper part of the abdomen.

6. It is suggested that this may have some bearing on the frequency with which postoperative pulmonary complications occur after operations in this region.

7. Such complications produce a secondary reduction in vital capacity and may be predicted by this means before clinical symptoms appear.

8. Acute inflammatory lesions and postoperative complications within the abdomen also cause a reduction in vital capacity.

9. The administration of a general anesthetic does not have any appreciable effect.

10. Accurately controlled observations on the vital capacity of surgical patients before and after operation serve as a satisfactory criterion of their general postoperative condition.

ASCARIASIS OF THE GALLBLADDER

LITERATURE AND CASE REPORT

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Ascariasis is not usually grouped among the diseases requiring surgical intervention, and its presence in what may be called a surgical form is probably seldom diagnosed prior to operation. Ascariasis of the gallbladder is especially unusual, if one may judge by the infrequency with which it has been recorded in the literature. The finding of an ascarid in the gallbladder of a woman on whom I recently performed an operation for cholecystitis suggested this study.

LITERATURE

The literature contains numerous references to cases in which ascarids were found in other unusual sites: the urinary passages, the appendix, the pancreatic duct, the common bile duct and the hepatic ducts. Ascarids in these locations were credited with causing many types of symptoms, chiefly those of obstruction and inflammation, presenting, however, few if any pathognomonic peculiarities. DaCosta [1] in his textbook on surgery, does not mention ascariasis among the diseases of the gallbladder. Only three direct references to the presence of parasitic worms in the gallbladder were found in the literature. Of the many other articles indexed under "ascariasis," "gallbladder," and other heads, only the more likely were investigated. In this group, only five additional articles were found in which ascariasis of the gallbladder was mentioned.

In 1765, Lorry,[2] quoted by Aviles, recorded the first case found in the literature. He cited the case of an insane patient in whose gallbladder three ascarids were found at necropsy. This patient vomited a worm prior to death and died in convulsions, presumably caused by the presence of the parasites in the gallbladder. Aviles [3] recorded a case of ascariasis of the gallbladder encountered at operation and summarized

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1. DaCosta, J. C.: Modern Surgery, ed. 9, Philadelphia, W. B. Saunders Company, 1925.

2. Lorry, A. C.: De melancholia et morbis melancholicis **16**:339; **8**:429, Lutetiæ Parisiorum. P. G. Cavelier, 1765.

3. Aviles, J.: The Rôle of Ascariasis in Gallbladder Disease, Surg. Gynec. Obst. **27**:459, 1918.

the literature. He quoted two other authors, Bonfils [4] (1858) and Devaine [5] (1860), who had recorded "a few cases." Bonfils quoted Lorry, Devaine quoted Lorry's case and two other cases which were attributed to Heaviside and Bloch.

In 1922, Butt [6] added a case of his own to the literature and quoted Tyau [7] (1920) and Eberle [8] (1920), who reported two more cases. Up to the time of this writing no other cases of ascariasis of the gallbladder were found in the literature. However, Benedict,[9] in 1926, reported a case in which *Taenia saginata* was found in the gallbladder. Because of its similarity to the cases in which *Ascaris lumbricoides* was present in the gallbladder, it is probably of more than passing interest.

That animals as well as man may harbor ascarids in the gallbladder was ascertained from Schlotthauer.[10] He, dealing with numerous laboratory and domestic animals, stated that he had occasionally encountered ascarids in the gallbladder of swine. They were apparently an incidental observation, and no symptoms or pathologic changes were ascribed to their presence.

Aviles called attention to the wide and almost universal geographic distribution of *Ascaris lumbricoides,* and said that the pathogenicity of the parasite depended on three characteristics. The first was a poisonous material found in extracts of the worm; the second was the carrying of infection by the motile and migratory traits of the ascarid, and the third was mechanical, the obstruction in a hollow viscus sometimes caused by a parasite or group of parasites. The last two characteristics are of primary importance in considering ascariasis of the gallbladder.

Another important consideration is the life cycle of the parasite. Knowledge of this is necessary in attempting to determine whether an ascarid found in the gallbladder has come there as an ovum and subsequently hatched, or whether its presence there is the result of migration of a worm from the intestine. Ova of *Ascaris lumbricoides* were found in the gallbladder and bile ducts by Tyau, and he also found

4. Bonfils, E. A.: Des lésions et des phénomènes pathologiques determinés par la presence des vers ascarides lombricoides dans les canaux biliaires, p. 28, Paris, Rignoux, 1858; reprinted from Arch. gén. de med., Paris, 1858.

5. Devaine, C.: Traité des entozoaires et des maladies vermineuses de l'homme et des animaux domestiques, Paris, 1860, p. 156.

6. Butt, A. P.: Round Worm (Ascaris lumbricoides) in Gallbladder, Surg. Gynec. Obst. **35**:215, 1922.

7. Tyau, E. S.: Pathogenicity of Ascaris lumbricoides; Report of Cases, M. J. China, **6**:21, 1920; abstr., J. A. M. A. **75**:135 (July 3) 1920.

8. Eberle, D.: Ascarids in the Liver and Pancreas, Schweiz. med. Wchnschr. **50**:1110, 1920; abstr., J. A. M. A. **76**:344 (Jan. 22) 1921.

9. Benedict, E. B.: Taenia Saginata in the Gallbladder, J. A. M. A. **87**:1917 (Dec. 4) 1926.

10. Schlotthauer, C. S.: Personal communication to the author.

worms as short as 1¼ inches (3.1 cm.) in length in the bile ducts. Stiles[11] said: "The eggs of the ascaris will not hatch as long as they are in the human body. They must pass out and there undergo their changes. The young worm is developed inside the egg, the egg is then swallowed, hatches out in the stomach, crawls through the wall of the stomach, gets into the circulation, goes to the heart and lungs, up the trachea, down through the stomach to the small intestine, and there develops further." Others differ with this opinion and believe that ova which contain embryos and which are still surrounded with the mulberry envelop will develop into roundworms in the human intestine.

The cases of ascariasis of the gallbladder were not recorded in anything approaching a uniform manner. There are, however, certain similarities in all of them which form a rather clearcut composite picture.

The data in the two cases quoted by Devaine were very meager and the brief facts of Lorry's case have already been quoted. The cases of ascariasis reported by Aviles, Tyau, Eberle, and Butt and the case of taeniasis reported by Benedict were complete enough for instructive study, and I shall summarize them.

REPORT OF CASES

AVILES' CASE.—A woman, aged 38, had suffered with attacks of pain in the upper part of the right side of the abdomen for twenty-six years. Attacks had been increasingly severe for the last year. She had occasionally vomited roundworms. For six days before admission to the hospital she suffered continuous pain, which radiated to the right shoulder blade and was accompanied by slight icterus and moderate fever. The diagnosis of cholecystitis with probable cholelithiasis was made, and the abdomen was explored. An *Ascaris lumbricoides* 8 inches (20.32 cm.) in length was found in the gallbladder obstructing the cystic duct. The worm was removed, and a cholecystostomy was performed. Anthelmintics were administered during the postoperative convalescence.

TYAUS' CASE.—A man, age not mentioned, had suffered for three months with epigastric pain which radiated to the liver region but not to the shoulder. A mass suspected of being the gallbladder was palpable. Ova of *Ascaris lumbricoides* and *Trichuris trichiura* were found in the stools. Santonin was administered and eighteen roundworms evacuated, but the symptoms were not relieved. Five days later, the patient died. Necropsy disclosed nine roundworms occluding the cystic duct. There were multiple abscesses of the liver and several roundworms in the hepatic ducts. Ova were found in the gallbladder, the biliary ducts and the liver itself.

EBERLES' CASE.—A boy, aged 9 years, had been treated with santonin for intestinal parasites. Five months later, he complained of pain in the upper part of the right side of the abdomen, and there were symptoms of jaundice. The abdomen was explored for gallbladder disease, and a living roundworm was found in the gallbladder. Three more worms, all alive, were found in the hepatic duct.

BUTT'S CASE.—A woman, aged 30, mother of three children, had had six attacks of severe pain in the upper part of the right side of the abdomen during

11. Stiles, C. W.: Personal communication to Butts.

two years. She had frequently passed roundworms in her stools. When seen she had suffered pain for six days, and a diagnosis of cholecystitis with probable cholelithiasis was made. Operation disclosed a distended but otherwise rather normal looking gallbladder. It was incised, and a roundworm about 6 inches (15.24 cm.) in length was removed. No stones were found. The incision in the gallbladder was closed, and the abdomen was closed without drainage.

In the next case, *Taenia saginata* was found in the gallbladder. This case is summarized because of its similarity to the cases of ascarasis of the gallbladder and because of the great length attained by the worm.

BENEDICT'S CASE.—A man, aged 74, had had several previous attacks of pain in the upper part of the right side of the abdomen which were not accompanied by jaundice. The patient had known for two years that he harbored a tapeworm, and he remembered eating morsels of raw beef when he was a butcher, fifty years before. Ten days after an operation for cataract, he was seized with severe pain in the upper part of the abdomen, and a diagnosis of acute cholecystitis was made. The abdomen was opened, and three gallstones and *Taenia saginata* 10 feet and 2 inches (310 cm.) in length were found in the gallbladder. Cholecystostomy was performed.

Aviles, from his study of the subject, concluded that such cases are rare, and that the diagnosis is not easy. He pointed out that the vomiting of an ascarid complicating an attack of gallbladder colic should make one suspect the possibility of ascariasis of the gallbladder and is an indication for exploration of the abdomen. Furthermore, he strongly advised the use of anthelmintics as a prophylactic and postoperative measure. Butt concluded that cholecystectomy is probably the treatment of choice in view of the fact that ova might remain in the gallbladder and lead to a recurrence of the disease.

AUTHOR'S CASE.—B. N., aged 45, the wife of a Virginia farmer, entered the University of Virginia Hospital on Nov. 12, 1927. Her family history was not important, and her past history was essentially negative. She had had five pregnancies with three miscarriages, but her health had been consistently good until the onset of her present trouble.

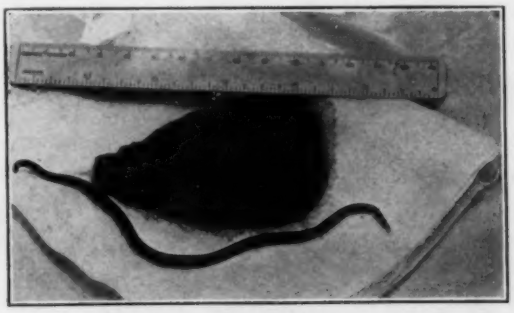
On admission she complained that she had suffered with pain and soreness in the upper part of the abdomen for five weeks. Her illness was initiated by a "cold chill" which occurred while she was milking. She fainted, but recovered consciousness in a few moments and was put to bed complaining of a severe, knifelike pain in the upper part of the right side of the abdomen, radiating to the right shoulder blade. After a few hours, the pain partially subsided, but she had marked residual soreness and tenderness along the costal margin on the right side. These continued throughout the five weeks, and occasionally there were exacerbations of pain. There was not any nausea, vomiting or jaundice and no clay stools. The patient was confined to bed the greater part of the time.

Examination of the patient disclosed adiposity of moderate degree, dental caries, diseased tonsils and well marked tenderness and muscle spasm over the gallbladder region. Jaundice was not present; temperature was 99.4 F., and the leukocytes numbered 8,800. Routine blood count, urinalysis and Wassermann tests of the blood were all negative. Roentgenograms of the gallbladder

following intravenous injections of dye (tetra-iodo-phenolphthalein) failed to show any trace of a shadow of the gallbladder. Subacute cholecystitis with probable cholelithiasis was the diagnosis made, and an operation was advised.

On Nov. 17, 1927, I made a high incision through the right rectus muscle and explored the abdomen. The pelvic organs, appendix, kidneys, stomach and duodenum apparently were normal. The gallbladder was surrounded with recent adhesions to adjacent structures and was distended to a diameter of about 8 cm. at the fundus. Stones were not palpated, and the gallbladder did not empty under pressure. The walls were thickened. The common bile duct was not enlarged, and stones were not palpated in it. The gallbladder was removed from below outward, and the abdomen closed without drainage.

Pathologic examination of the gallbladder disclosed thickening and subacute inflammation of the walls. The mucosa showed some cholesterin deposits. The bile was of about normal consistency. In the gallbladder was discovered a living, actively motile roundworm, *Ascaris lumbricoides*, which measured 17.5 cm. in length (fig. 1).



Ascarid and gallbladder removed from patient.

Postoperative convalescence was quite uneventful, and the patient was allowed to go home on Dec. 4, 1927, eighteen days after her operation. Anthelmintics were advised to rid her of any parasites remaining in the intestinal tract.

Following the unusual and unexpected operative observation, the patient was carefully questioned concerning her previous history. She again denied any former attacks of pain in the gallbladder region, and said that she did not know that she harbored intestinal parasites until the day before operation. On that day she was nauseated and vomited an *Ascaris lumbricoides* 15 cm. in length.

COMMENT

The problem of how a parasitic worm may have arrived in the gallbladder is one difficult to solve. One must assume either the hatching of an ovum in the gallbladder or the migration from the intestine of a worm sufficiently small to pass through the cystic duct. In Benedict's case, a taenia 10 feet (304.8 cm.) long was found in the gallbladder. How long this parasite had been in the gallbladder is not easy to determine, although it is improbable if not entirely impossible to suppose that it could have passed through the cystic duct while measuring anything approximating 10 feet in length.

Probably the preoperative diagnosis of ascariasis of the gallbladder will never be made with any great degree of certainty. In the first place, it is obviously a rather rare condition and one which would hardly be thought of unless one were to bear it in mind in all cases of cholecystic disease. In the second place, the symptoms of the disease do not differ in general from those of the commoner varieties of inflammation and obstruction of the gallbladder and cystic duct.

Outline of Cases Found in Literature and of My Own Case

Case Reported by	Sex	Age	Past History: Ascariasis	Past History: Gallbladder	Complaint	Pre-operative Diagnosis	Observations at Operation	Results
Heaviside and Bloch, quoted by Devaine, 1860	2 cases, were not recorded in detail							
Lorry, 1765...	..	..	Vomited an ascarid	.....	Convulsions	.....	.....	Necropsy: three ascarids in gallbladder
Aviles, 1918...	F	38	Vomited ascarids occasionally	Periodic attacks for 26 years	Gallbladder colic for 6 days	Cholecystitis; probable cholelithiasis	Ascarid 8 inches long in gallbladder	Cholecystostomy
Tyau, 1920....	M	..	Ova found in stools	Pains for 3 months	Pain and mass in gallbladder area	.....	.....	Necropsy: nine ascarids in cystic duct
Eberle, 1920...	M	9	Treated for ascariasis 5 months previously	Pain and jaundice	Pain and jaundice	Cholecystitis	Ascarid in gallbladder	Cholecystostomy presumably
Butt, 1922.....	F	30	Frequently passed ascarids in stools	Periodic pain for 2 years	Gallbladder colic for 6 days	Cholecystitis; probable cholelithiasis	Ascarid 6 inches long in gallbladder	Gallbladder closed without drainage
Benedict, 1926	M	74	Known to have taenia 2 years; raw beef eaten 50 years before	Several attacks previously	Gallbladder colic for few hours	Acute cholecystitis	Taenia 10 ft. 2 in. long found in gallbladder	Cholecystostomy
Morton, 1928..	F	45	None except vomited ascarid day before operation	None	Gallbladder pains for 5 weeks	Cholecystitis; probable cholelithiasis	Ascarid 17.5 cm. long found in gallbladder	Cholecystectomy

The case reported herewith illustrates the point. The history and physical observations were similar to those in numberless other cases of ordinary cholecystic disease. The laboratory and cholecystographic data were equally commonplace. The diagnosis of cholecystitis with probable cholelithiasis seemed obvious.

There was, however, one fact not taken into consideration in making the diagnosis. That was the vomiting of the ascarid the day before operation, ignored as not important at the time and yet in the light of subsequent observations it proved to be one of the most significant features of the case.

I made a tabulation of the cases of ascariasis found in the literature and included my own case and that of Benedict, in which the parasite was not an ascarid but *Taenia saginata*. The incidence in the two sexes is about equal, and the age limits vary between 9 and 74 years. There are two factors, however, which are constant in every case; the history of intestinal infestation with parasites and symptoms referable to the gallbladder.

These two constant features of the cases do not assure the diagnosis, but when elicited they should at least lead one to bear in mind the possibility of ascariasis of the gallbladder.

The treatment for ascariasis of the gallbladder is the same as that for inflammation or obstruction of the organ due to other causes. As suggested by Butt, cholecystectomy is probably the operation of choice. Anthelmintics are indicated as a prophylactic measure in all persons with intestinal parasites and as a postoperative measure in ascariasis of the gallbladder to rid the patient of any remaining parasites.

SUMMARY

The literature was searched for cases of ascariasis of the gallbladder. Seven cases were found and five have been briefly summarized. A case of taeniasis of the gallbladder was found and also summarized. It was similar in many respects to the cases of ascariasis. All cases had been diagnosed only after operation or necropsy.

A case of ascariasis of the gallbladder which I encountered has been described, and the condition discussed in relation to its etiology, diagnosis and treatment. It was pointed out that the disease is rare and difficult to diagnose. Furthermore, the treatment is surgical and cholecystectomy is probably the operation of choice. Anthelmintics are indicated for prophylactic use and postoperatively. The history of ascariasis in a patient with signs of cholecystic disease should always suggest the possibility of ascariasis of the gallbladder.

A REVIEW OF UROLOGIC SURGERY

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(Continued from page 178)

URETER

Transplantation.—Peterson [25] believes that the extraperitoneal route is more desirable than the intra-abdominal in the transplantation of the ureters into the bowel. Even with the most skilful care, the wound may become infected, and tight approximation of the suture line is difficult. If urine or feces escape around the opening through the mucosa, the patient usually succumbs, unless the operation has been performed extraperitoneally. Technically, it is not more difficult since the ureters are readily exposed and easily transplanted.

Peterson reports several cases of incurable vesicovaginal fistula in which transplantation of the ureters was carried out in order to secure sphincteric urinary control. He does not expose the patient to the additional risk of transplanting both ureters at the same operation.

In Coffey's [26] earlier papers, he advises the insertion of a small rubber tube into the ureter before transplantation. The greatest trouble in ureteral transplants, especially in animal experimentation, is infection emanating from the opening in the intestine. At present, Coffey uses the following technic: The bowel is cleansed with castor oil and enemas before operation. The abdomen is opened low near the median line, and the sigmoid is pulled down into the field of operation. A symoidoscope is inserted into the rectum, and the lower part of the bowel is flushed by a pointed cannula inserted through the wall of the sigmoid. The rectum is then packed full of dry gauze, and the sigmoidoscope is removed.

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25. Peterson, Reuben: Transplantation of the Ureters into Bowel to Secure Sphincteric Urinary Control in Incurable Vesicovaginal Fistula, Am. J. Obst. & Gynec. **14**:492, 1927.

26. Coffey, R. C.: Completed Aseptic Technique for Implantation of Ureter into Large Bowel, Surg. Gynec. & Obst. **45**:816, 1927.

The ureters are located and severed between small artery forceps as near to the bladder as possible. The distal stump is cauterized with phenol and ligated. The proximal end of the ureter is liberated for 7.5 or 10 cm. by slitting the peritoneum over it. Ureteral catheters, provided with rubber cuffs to prevent slipping, are inserted into the ureter. A strong linen thread is passed twice around the ureter and cuff and tied tightly with a double or surgeon's knot. A similar suture is then tied around the ureter below the incision to prevent infection ascending the ureter from the bowel. Urine usually begins to escape from the catheters at once.

To avoid too much narrowing, one incision should be higher in the bowel than the other. The incision should begin near the mesenteric edge and extend downward and obliquely toward the antemesenteric border to avoid as many of the large vessels as possible. It should be about 3.8 cm. in length, and through the peritoneum and muscle, thus permitting the mucosa to pouch out partially. A small stab wound is made in the mucosa between traction sutures. A little of the gauze already inserted in the rectum is picked up with mosquito forceps, and to this the end of the catheter is attached. After both of the catheters are attached to the gauze, the gauze is gradually withdrawn from the rectum and in so doing the ureteral ends are drawn into the openings in the mucosa. The catheters must be marked to indicate the right and left kidney. After the ureter is drawn past the ligature, the free ends of the two traction loops are tied across the ureter to close the opening partially. These traction loops, inserted before the mucosal puncture, are important in handling the intestine.

[ED. NOTE.—Coffey's method of ureteral transplantation is, at the present time, accepted by most urologists as the one giving the most satisfactory functional results. Not all urologists agree as to the indications for ureteral transplantation. Hinman [27] believes in transplanting the ureters into the bowel in a small group of cases. Usually this method is employed now only as a last resort, but if freed from some of its dangers, it could be applied to a larger number of cases. Hinman believes that a surgical victory over this defect must include restoration of natural relationships. To insure satisfactory renal function, a first stage preparatory nephrostomy should be performed; this is followed subsequently by ureteral transplantation.]

Marion [28] states that ureteral transplantation is of doubtful value. If it is performed, it is necessary to be ready to operate on the corresponding kidney in case of complications. In some cases, instead of

27. Hinman, Frank: The Indication of Nephrostomy Preliminary to Ureterorectoneostomy, J. A. M. A. **86**:921 (March 27) 1926.

28. Marion, M.: Suite de la discussion sur les implantations urétérales, J. d'urol. méd. et chir. **20**:58, 1925.

reimplantation, primary or secondary nephrectomy may be desirable. He believes that in most cases the kidney of the ureter that has been transplanted becomes dilated and gradually atrophies. Legueu, in discussing Marion's statements, states that he has noted only one favorable case of transplantation reported in the literature, that of a patient who, seventeen years after transplantation of the ureter, still had a well functioning kidney.

Papin [29] states that in his experience, ureteral transplantation is more satisfactory than simple nephrostomy. Isolation of the ureter from its normal connections disturbs its functioning power; it becomes atonic and dilated and contracts more slowly and less frequently. All surgical procedures on the ureter cause infection; the resulting dilatation of the ureter is responsible for most ascending infections. Papin gives three methods of transplanting the ureter: transplantation to the skin, transplantation into the urethra and transplantation into the intestine. He believes that the transplantation to the skin is the safest of the three methods.

Smitten [30] compiled a series of 318 cases of implantation of the ureters into the intestine with the following results: fistula of the bladder, 156 cases, with death in 34 (21.8 per cent); anomalous conditions, 58 cases, with death in 18 (31 per cent); malignant tumors, 98 cases, with death in 61 (63 per cent), and inflammatory processes, 4 cases, with death in 3 (75 per cent). Of the total number of patients, 116 died and 200 recovered; in 2 cases the result was not known. Thirty patients died from peritonitis, 21 from urinary infection, 5 from pneumonia and 3 from tuberculosis. The late results, from six months to ten years, were known in 71 cases. In 6 of these death was caused by renal insufficiency, in 3 by recurrence of the original condition, and in 2, by intercurrent disease.

In many cases, the time consumed and the difficulty of the operation do not justify the attempt to preserve renal tissue. The ureter is often already dilated and infected and the corresponding kidney is only slightly infected. On the other hand, in certain cases, such as exstrophy of the bladder in young patients, ureteral transplantation is unquestionably a life-saving procedure.

Regurgitation.—Graves,[31] in continuing his studies on regurgitation from the bladder into the ureters under normal conditions at the ureterovesical juncture, investigated the pressure curve of the bladder

29. Papin, M.: La derivation haute des urines, Presse méd. **2**:1412, 1925.

30. Smitten, A. T.: Ueber die Einpflanzung der Ureteren in den Darm, abstr. Ztschr. f. urol. Chir. **19**:219, 1926.

31. Graves, R. C.: IV. Studies on the Ureter and Bladder with Especial Reference to Regurgitation of Vesical Contents; Bladder Pressure-Curve in Human, J. Urol. **18**:321, 1927.

in man. The fundamental requisite is a sustained tonic contraction of the musculature of the bladder as it actively resists distention. Atonic bladders never regurgitate; postmortem experiments are futile. Moreover, as Graves has shown, there are no intrinsic anatomic differences, such as have been claimed, between the ureterovesical relationship of the laboratory animal and of the human being.

In general, then, it may be stated that there are two groups of bladders so far as reaction to distention is concerned. One group, marked by an active state of muscle tone, resists distention, and in these tracings of intravesical pressure show a constant, relatively rapid rise of tension as filling proceeds. Such bladders will regurgitate when the normal outlet is obstructed in a large percentage of cases, as he has demonstrated in a long series of observations on animals. The other group, in which tone is relatively lacking, consists of bladders that offer feeble or no resistance to the force of distention. These bladders fall away from the distending fluid, and there is no appreciable sustained rise in intravesical pressure until the end-stage of filling is reached, when, as has been said, a purely mechanical tension is produced because the sac can hold no more. In this passive type of bladder, regurgitation does not occur.

As Graves was dealing with fundamental reactions of smooth muscle, it seemed reasonable to expect in starting this particular investigation that the bladder of man, in its behavior during distention, would fall into one or the other of these groups and so give further evidence of support or denial of the clinical theory of regurgitation. To answer this phase of the question, he has made use of an instrument which has just been completed and which makes it possible to record accurately the development of intravesical pressure during filling of the bladder. The instrument consists of a reservoir from which the bladder may be filled with a definite volume and rate of speed through the action of a small piston pump. There is also a mercury manometer which communicates with the recording device and which is in open communication with the cavity of the bladder throughout the period of filling. Geared to the pump is a small kymograph which provides the permanent record of the manometer as it registers the changes in intravesical tension.

Thus far, however, he has noted only one case, that of a long-standing stricture with infection, which would not be classified beyond question with the active tonic group of bladders that he has described experimentally and that permitted reflux in so large a percentage of cases. Only the atonic bladder of disease of the spinal cord was of the passive type. The curves for the human being are similar to those for animals; from this analogy, Garves claims support for the probability of regurgitation in the bladder of man.

PROSTATE

Hypertrophy.—Troell [32] reports his results in a series of 93 cases in which prostatectomy was performed, with 5.4 per cent primary mortality (82 cases of benign hypertrophy of the prostate with 4.9 per cent mortality and 11 cases of prostatic carcinoma with 9 per cent mortality). Troell accentuates the need for preoperative treatment, especially drainage of the bladder.

Four and three-tenths per cent of the patients had epididymitis; the occurrence of this condition can be limited by a frequent change of the urethral catheter both before and after operation. In the cases of carcinoma, permanent results following prostatectomy were not good; in some cases, recurrence and metastasis occurred six months after operation. Troell believes that metastasis to bone may be favorably influenced by repeated treatments with the roentgen rays.

Wildbolz [33] gives the following indications for prostatectomy: (1) permanent retention of a considerable quantity of urine in the bladder (from 150 to 200 cc. or more); (2) frequent attacks of complete retention; (3) long-standing infection of the bladder, and (4) severe repeated hemorrhage from the hypertrophied prostate.

He does not perform prostatectomy as a prophylactic measure. It is never a harmless procedure and is justified only when the patient is in real danger from disease. Wildbolz considers the water test one of the most important of all the tests for renal function. Suter of Basle refuses to perform an operation unless the urine concentrates to a specific gravity of at least 1.017. Lehmann considers 1.018 and Rubritius 1.015 to be the minimum. Wildbolz shows that these figures are unnecessarily high, as twenty-six of his patients recovered in spite of a much lower concentration of the urine. In eighteen, it was from 1.011 to 1.014; in seven, it was 1.010, and in one 1.009.

Negro and Colombet of Marion's Clinic state that operation should not be performed unless at least 42 per cent of phenolsulphonphthalein is eliminated in the first hour. Blanc considers 55 per cent as a minimum. The majority of Wildbolz's patients eliminated more than 30 per cent of phenolsulphonphthalein in the first hour, but thirteen eliminated from only 20 to 30 per cent and ten less than 20 per cent.

The observations of Wildbolz and other investigators show that prostatectomy will be followed by uremia if the urea of the blood is more than 100 mg. per hundred cubic centimeters, and probably so if it is over 80 mg. When the blood contains from 50 to 80 mg., the operation

32. Troell, Abraham: Prostatectomy: Some Remarks About Indications, Technique and Results, Acta chir. Scandinav. **62**:133, 1927.

33. Wildbolz, Hans.: Indication and Execution of Prostatectomy, Proc. Roy. Soc. Med., Sect. Urol. **20**:50, 1927.

is always dangerous and should be performed only when other tests of renal function give good results.

Suitable preoperative treatment usually results in reestablishing renal function and in rendering the patient fit for prostatectomy. In some cases, the function of the kidney cannot be improved sufficiently by catheter drainage; in these, the two-stage operation should be performed.

Wildbolz believes that the perineal operation puts much less strain on the heart and lungs and causes less shock than the suprapubic. He finds that suprapubic prostatectomy is usually followed by an increase in the blood urea, lasting for several days, but after a perineal operation there is practically no such increase. The perineal operation does not cause nearly so much bleeding, and the perineal wound does not hinder respiration or expectoration. Furthermore, the perineal operation is not followed by necrosis of the tissues, and the wound is so well drained that there is only a slight amount of absorption which may increase the blood urea.

Wildbolz uses the same incision in performing perineal prostatectomy as that employed by Zuckerkandl, Albarran and Proust, with a slight modification used also by Young. The central tendon of the perineum is exposed and cleaned. A small transverse incision is made in it close to the bulb, and the recto-urethralis muscle is exposed. This is divided close to the urethra, and the prostate is exposed by blunt dissection. On the anterior wall of the wound are the bulb and transversus perinei muscle, and on the posterior side lies the rectum. The prostate is pressed down toward the perineum by a metal catheter introduced into the urethra. It is covered by the fascia of Dénonvillier. This fascia is divided transversely just above the apex of the gland and is pushed backward, exposing the posterior portion of the capsule in the wound. Wildbolz makes a vertical incision in the median line, commencing at least 1 cm. above the apex, which involves only the capsule of the prostate and does not penetrate to the urethra, and which enables one to distinguish between capsule and adenomatous masses. Both lobes of the prostate are enucleated as far as possible. The prostatic urethra is divided transversely just at the lower end of the adenomatous mass, the upper end still being left in connection with the neck of the bladder. Young's retractor is introduced through the prostatic urethra into the bladder, and the upper end of the urethra is divided close to the neck of the bladder, with any adhesions still existing between the adenoma and the capsule of the gland. The bladder is washed out to remove blood clots.

Four sutures are placed through the neck of the bladder, and afterward through the stump of the urethra at the apex of the gland. They unite the neck of the bladder to the urethra inside the prostatic cavity. Thus this procedure restores normal anatomic conditions as far as possi-

ble. Before the sutures are tied, the metal catheter is replaced by a silk one which is retained for twelve or fourteen days after operation. A drainage tube is placed in the opening of the prostatic capsule, which is partially closed by means of a single suture. Packing is not inserted. The superficial part of the wound is brought together by a few sutures. As a rule, the wound heals by primary intention. The perineal drainage tube is removed three or four days after operation, and the fistula heals so quickly that the catheter can be removed in from ten to fourteen days. The patient is then able to pass urine normally through the urethra.

The main points in which this method differs from other perineal operations are: (1) complete resection of the part of the prostatic urethra encircled by the adenoma, (2) suture of the neck of the bladder to the urethral stump, and (3) suture of the incised prostatic capsule.

This method of prostatectomy causes only slight injury to the tissue. Little blood is lost, and serious postoperative hemorrhage rarely occurs. Infection rarely follows perineal prostatectomy, the wound is smooth, there are no cavities or flaps of necrotic tissue, and the area is well drained.

Mortality after perineal prostatectomy is lower than after suprapubic. Wildbolz reports a mortality of 15 per cent in 105 suprapubic operations in contrast to a mortality of only 6.5 per cent in 305 perineal operations. In 29 of Wildbolz' cases, healing of the perineal fistula was delayed. In 1 case, the fistula persisted for more than three months. In 3 cases, there was a persistent urethrorectal fistula which required another operation for relief. Partial incontinence lasting for a few weeks was observed in 6 cases, and in 5 others it persisted for more than four months.

In discussing indications for prostatectomy, Hunt[34] says that inability to empty the bladder and persisting residual urine are the outstanding and important indications for prostatectomy. The absence of residual urine, however, does not constitute contraindication to operation if the gland is enlarged and there are subjective symptoms. With any amount of residual urine as a result of prostatic obstruction, the indications for operation are clear; with frequency, difficulty, pain and other symptoms, but with slight enlargement of the gland and no residual urine, the advisability of operation is questionable.

Death following prostatectomy has been due to the following causes, in order of frequency: (1) coexisting or preexisting cardiovascular, renal and pulmonary diseases; (2) surgical accidents, such as hemorrhage, and (3) postoperative complications. In recent years, effort has been directed toward providing factors of safety which tend to eliminate

34. Hunt, V. C.: Immediate and End Results of Suprapubic Prostatectomy: A Consideration of the Factors Involved, Canad. M. A. J. **17**:1467, 1927.

these causes of death. Two types of cases present the greatest hazards in prostatectomy: those in which prolonged urinary retention has resulted in marked renal insufficiency, and acute uremia and death are readily precipitated by immediate prostatectomy; and those in which the urinary tract is free from infection, only a few ounces of urine are retained as the result of prostatic obstruction, and there is little or no impairment of renal function, a type of case in which general sepsis is likely to be precipitated by immediate prostatectomy. Experience has taught that in the first type the patient may recover from actual or potential uremia, that renal efficiency may be restored or approach normal, and that the cardiovascular renal reserve may be increased by a period of careful and adequate preoperative treatment. In the second type of case, the patient may likewise be provided with immunity to infection by the same means.

Recent investigation of the relationship of preliminary treatment to mortality following prostatectomy has definitely established the necessity of preliminary treatment in all cases.

Hunt believes that in 75 per cent of cases prostatectomy can safely be performed by the one-stage visualized operation, which permits the application of the general principles of surgery, that is, exposure, accuracy of conduct and complete hemostasis. He emphasizes the importance of avoiding anesthesia by inhalation, and of using the various forms of local anesthesia. The advantages of the one-stage over the two-stage operation are also dealt with, and the operation is illustrated.

The discussion of mortality in this paper is based on a five year period, during which time suprapubic prostatectomy was performed on 995 patients at the Mayo Clinic.

This series has been carefully reviewed with regard to the mortality rate, subsequent course and ultimate function. In several series of cases of 100 each, there were no deaths. The mortality rate by years, however, has varied from 6.4 per cent in one year to 2.4 per cent in 1925, with an average rate of 4.2 per cent for the five year period. In this connection, it should be stated that previous to January, 1925, preoperative preparation was carried out in only about 75 per cent of the cases. The remaining 25 per cent of the patients were in excellent general condition and were operated on without preoperative preparation. The effect of preoperative preparation in all cases after January, 1925, became apparent immediately.

The one-stage operation was performed in 783 cases (78.6 per cent) of the series with 34 deaths, a mortality rate of 4.3 per cent. In 212 cases, the two-stage operation was performed with 8 deaths, a mortality rate of 3.7 per cent. This is in contrast with a previous report on an earlier series of cases in which the mortality rate of the two-stage operation was one and a half times as high as that for the one-stage operation.

The causes of the 42 deaths were as follows: uremia and renal insufficiency, 10; pulmonary embolism, 9; general sepsis, 7; cardiovascular disease, 6; secondary bleeding, 4; pneumonia, 3, and bleeding duodenal ulcer, general peritonitis, and erysipelas, 1 each. Renal insufficiency and uremia are important factors in cases in which urinary obstruction and retention have existed for a long time. Pulmonary embolism has not occurred as a cause of death since the adoption of preoperative preparation of all patients. It has continued to be difficult to combat general sepsis, which usually emanates from an acute exacerbation of cysto-ureteropyelonephritis. It would seem that prostatectomy had little to do with some of the deaths, but these deaths are included in the surgical mortality.

The late results were good. The preoperative difficulty in urination was relieved in 95 per cent of the patients.

Ware [35] condemns the packing of the prostatic capsule with gauze tampons, as ineffective and cumbersome. The Hagner and Pilcher bags overcome the disadvantages of gauze tampons, but these bags come in contact with the prostatic bed by drawing taut the rubber tube at its exit from the urethra and maintaining this pull by anchorage to a cradle with its points of support on the pelvis. These cradles are at times difficult to adjust and hamper the movements of the patient. Ware has devised a method which he believes is superior. On completion of enucleation, a Nélaton catheter is introduced until the vesical end becomes visible or can be palpated. A length of water-proofed linen fishing line is secured to the vesical end drawn into the wound. The catheter is withdrawn until a length of thread emerges which passes over a pulley at the foot of the bed and reaches the floor. Some Penrose tubing is secured to the thread emerging from the abdominovesical wound by throwing a knot about loosely coiled folds of the tubing arranged in roset fashion, or with greater refinement transfixing loops of tubing with the same thread armed with a large needle. When this is accomplished, the thread coursing through the urethra is pulled on, causing the rubber tampon to follow until the resistance of the prostatic bed at the introitus of the bladder is felt. Traction is sustained by applying a sand bag weighing from 3 to 5 pounds (1.4 to 2.3 Kg.) to the end of the thread, which is allowed to fall over the end of the operating table. When the patient is put to bed, the end of the thread, with its weight, plays over a pulley. The weight may be adjusted to the comfort of the patient. A thread attached to the tampon is allowed to emerge from the suprapubic wound at the time of closure.

In from forty-eight to seventy-two hours, when the need for hemostasis is past, the thread emerging from the urethra is cut close to

35. Ware, M. W.: Sustained Counterweight-Traction in Haemostasis of Prostatectomy, Ann. Surg. **86**:561, 1927.

the orifice, and the tampon is removed by pulling gently on the thread emerging from the wound. In removing the tampon, the coils of Penrose tubing are gently manipulated, and these tend to unfold themselves. leading to little discomfort.

[ED. NOTE.—The use of coiled Penrose tubing as a tampon is perhaps a new feature. One can imagine that it would be difficult in all cases to get such a tampon to fit snugly enough for hemostasis at all points of the capsule. The pull of a thread through the urethra also would possibly be associated at times with cutting into the urethral tissue at the region of the bulb or triangular ligament and might call for a considerable amount of adjustment. The principle of counterweight traction is not without virtue, although the attachment of the weight to a Hagner-Pilcher bag would seem more likely to produce less discomfort, hemostasis would be better because of more uniform contact of the bag with the prostatic capsule, and the removal of the bag might be more certain and less difficult as a rule than that of coils of Penrose tubing. Any type of persistent pull on the remaining muscles at the neck of the bladder tends to increase the possibility of transient incontinence. The system of weights suggested by Ware would at least offer consistent tension and be free from the irregularly strong and weak tension occurring when the urethral tube of the hemostatic bags is attached to the patient.]

Thomas [36] states that according to estimates, 50 per cent of patients with prostatic disease die within five years after the onset of obstruction, and that catheter life shortens this to two and a half years.

The function of the kidney is one of the most important factors in determining the qualifications of a patient for operation. An estimation of the urea nitrogen in the blood and the quantitative percentage output of phenolsulphonphthalein are the most commonly used tests of renal function. Normal values of urea nitrogen in the blood range from 12 to 20 mg. per hundred cubic centimeters; safe values range from 20 to 30 mg.; values of above 30 mg. should be regarded as dangerous, if not prohibitive of operation. The cardiovascular system and the blood are next to renal function in importance. As a result of the study on the cause of death following prostatectomy, Thomas notes the following rules: In cases in which low tension is present, when the systolic blood pressure is 110 or less, the diastolic must be more than 60; if the diastolic pressure is less than 60, the systolic must be more than 110. In cases in which high tension is present, when the systolic pressure is 180 or more, the diastolic must be less than 100; if the diastolic pressure is more than 100, the systolic must not be more than

36. Thomas, B. A.: Vital Factors in the Management of Prostatic Obstruction, Ann. Surg. **86**:563, 1927.

175. This is not so-called "pulse pressure" in its usual sense, but rather pulse pressure with systolic and diastolic limitations. Thomas has performed operations successfully in cases in which the systolic pressure was less than 110, or even 100, but the diastolic was always more than 60; conversely, he has had cases in which the diastolic pressure was less than 60, but the systolic was always more than 110. On the other hand, if the systolic pressure is more than 190, operation is a hazardous procedure, but it has been performed successfully several times when the diastolic pressure was less than 100; conversely, in many cases in which the diastolic pressure was as high as 120, prostatectomy has been performed with recovery, but the systolic pressure was invariably less than 175.

Age, in itself, is never a vital factor in prohibiting operation on the prostate. Thomas believes that it is unwise to force old men out of bed too soon. The many complications incident to epididymitis, to phlebitis and to embolism will thereby be averted.

Lowsley and Harrah [37] report a case of complete retention of urine in a man, aged 32, due to solid tumefaction of the prostate gland. Suprapubic cystostomy was performed.

The patient was subjected to suprapubic cystotomy under local anesthesia. Examination of the interior of the bladder revealed a rather firm mass about 4 cm. in diameter, extending from the floor of the neck of the bladder. A specimen was taken from this tumor-like projection and sent to the laboratory for examination. The bladder was drained by suprapubic suction for a period of fourteen days, after which the prostate was removed suprapubically under sacral and parasacral and regional anesthesia.

The growth had greatly increased in size since the first operation. Microscopic examination showed chronic inflammation suggestive of Hodgkin's lymphogranuloma.

Prostatitis.—Le Roy [38] reviews a series of thirty-four cases of suppuration in prostatic hypertrophy. He believes that these conditions are relatively common and often pass unnoticed, because at times the pain is only moderate, and pyuria, the patient's general condition, and the absence of unusual symptoms are not suggestive of the principal disease. Prostatic suppuration calls for energetic and immediate treatment; otherwise serious complications set in.

The urine is often clear, and, unless a rectal examination is made, the diagnosis may not be apparent until the condition is far advanced. The patient complains of a heavy feeling in the perineum and dysuria.

37. Lowsley, O. S., and Harrah, F. W.: Enlargement of the Prostate Gland with Characteristics Resembling Hodgkins' Disease, Ann. Surg. **86**:556, 1927.

38. Le Roy, C. M.: Des suppurations locales dans l'hypertrophie de la prostate, J. d'urol. méd. et chir. **23**:311, 1927; **23**:388, 1927.

There is a marked and rapid increase in the volume of the gland. The diagnosis is frequently confused with that of carcinoma of the prostate. A single lump on the surface of the prostate usually means the onset of malignancy. Numerous irregularities and lumps suggest prostatitis. Pus in the expressed prostatic secretion and the presence of eosinophilia help confirm a diagnosis of infection.

It is easier to make a diagnosis in acute cases, and these are more significant on account of the urgency of treatment. Prostatectomy is the procedure to be used, preferably in two stages. In acute forms, however, when the condition of the patient does not permit delaying operation, perineotomy must be performed to permit evacuation of the pus. At the same time, the bladder should be drained.

Casariego [39] advises perineal prostatotomy as the treatment for patients with urinary retention due to acute gonorrheal prostatitis; the posterior surface of the prostate is exposed by means of a deep longitudinal incision over both lobes. Persistence of retention of urine in a case of acute gonorrheal prostatitis is a sign of formation of abscess with more or less destruction of the gland. Prostatotomy avoids this and prevents the formation of caverns and fistulas of the prostate. It may often be considered as prophylactic treatment of chronic prostatitis.

Calculi.—Thomas and Robert [40] give an extensive review of the literature on prostatic calculi and report three cases. Although cases have been reported occasionally in the older literature, it usually was not considered that the stones originated in the gland itself. In more recent times, however, prostatic calculi have ceased to be a rarity; they are, in fact, much commoner than the number of reported cases would indicate. The etiologic factor of true prostatic calculi is still hypothetic. These calculi, whether septic or aseptic, have as a nucleus the corpora amylacea, which are albuminoid or nitrogenous-like bodies. They are usually multiple and the size of a "millet seed" or a "pea"; they are most common in middle and late life and are associated with other pathologic conditions. About 65 per cent do not cause symptoms and are found accidentally during routine roentgen-ray examination or at operation. A persistent and rebellious prostatitis should arouse suspicion of calculi. Serious sequelae, such as abscess and gangrene, are rare. Malignancy and inflammatory nodules can be differentiated in almost 100 per cent of cases by means of roentgenograms. Phleboliths must be taken into consideration. Surgical intervention by the perineal route is the procedure of choice, except in cases in which the calculus communicates with the urethra, when it may be removed through the urethroscope.

39. Casariego, A. G.: Prostatotomy as Treatment of Urinary Retention Due to Acute Gonorrheal Prostatitis, J. Urol. **18**:401, 1927.

40. Thomas, B. A., and Robert, J. T.: Prostatic Calculi, J. Urol. **18**:470, 1927.

BLADDER

Gangrene.—Carson [41] reviews 167 cases of necrosis and gangrene of the urinary bladder noted in the literature and added 6 cases in which necropsy was performed at the University of Maryland. Necrosis or gangrene of the bladder occurs as a result of: (1) interference with the circulation, internal or external pressure; (2) infection, general or local, with or without mechanical injury; (3) lesions of the central nervous system, and (4) chemical irritants.

There were 116 females and 57 males. The age varied from 3 to 75 years, the majority of patients being in the third and fourth decades.

Of the 173 patients, 102 died, 67 recovered and 4 were not traced. In 40 cases, the gangrene occurred in pregnant women; in 23 it occurred after labor; in 15 it was associated with retention of urine; in 11 females it was due to external pressure with retention in 6. Cystitis was the antecedent in 14 cases (7 males and 7 females). The cystitis was complicated by retention in 7 of the 14 cases by stricture of the male urethra in 9 cases, in which there were calculi in the urethra in 3. In 6 cases prostatic obstruction with retention was present, in 6 calculi in the bladder with retention, and in 6 irritants were a factor and retention was associated. General infections played a part in 15 cases, in 7 of which there was retention. In 12 cases there were lesions of the central nervous system, retention being present in 7. Trauma was the cause in 5 cases; retention was associated in 3 of these. Extroversion of the bladder through the urethra occurred in 5 cases. There were 20 miscellaneous cases, retention being an added factor in 10 of these. In all, retention was present in 76 (48.5 per cent).

Obstruction of the circulation will explain the cases of simple necrosis, but pathogenic bacteria must be present before putrefaction can take place.

In the 67 cases in which the patients recovered, the diagnosis was made by cystoscopic examination, suprapubic cystotomy or examination of the exudate passed by urethra.

At necropsy, the mucosa of the bladder was frequently found to be yellowish, greenish yellow, or black with a putrefactive odor, and the perivesicular tissues showed evidences of varying degrees of inflammatory reaction, trigonitis, peritonitis and ureteritis.

[ED. NOTE.—In view of the clinical rarity of gangrene of the bladder, Carson's excellent review is timely. From a pathologic standpoint, regardless of the exciting factor, one must feel that ultimate interference with the vesical blood supply to such an extent as to destroy nutrition is the real cause of the condition. This presupposes a fulminating infection, intense irritation, or severe trauma, as is borne out by the cases reported.]

41. Carson, W. J.: Gangrene of the Bladder, Ann. Surg. **85**:269, 1927.

Schüppel [42] states that cystitis gangraenosa dessicans is the most unfavorable form of inflammatory disease of the bladder. The name was given to this disease by Stöckel, who first described it and reported two cases. Although it is not possible to determine the number of reported cases, many have been observed.

The prognosis is grave. In Subotski's 167 cases, there were 90 deaths. Constantini reports 18 (40 per cent) deaths in a group of 45 women and 17 (51.5 per cent) in a group of 33 men. Houltani reports 8 (24.2 per cent) deaths among 33 pregnant women. This high mortality in women is explained by the association of this condition with unsuccessful abortion and adherent retroflexed uterus. The lower death rate among women in general is due to the fact that the urethra is shorter and permits more satisfactory drainage of the bladder. In men, prostatic disease, urethral stricture and tabes dorsalis are responsible for most of the deaths.

Successful treatment depends on an early diagnosis, which necessitates a study of the underlying causes. Frönstein classifies the cases into four groups:

1. The destructive factor began with some type of chemical which had been introduced into the bladder in too strong a concentration. In one case the condition was brought on by the introduction of 100 cc. of 20 per cent potassium permanganate, in another by the injection of 10 per cent sodium bromide for cystography.

2. In this group, the condition was caused by external pressure, such as retroflexion of the uterus, and urinary retention due to urethral stricture and prostatic hypertrophy.

3. The infectious cystitis had existed for a long time and gradually changed to a gangrenous type without mechanical factors.

4. In this group, the condition followed diphtheria, roentgen-ray therapy, impaction of calculus in the urethra, and extension from inflamed appendix.

The best treatment is the establishment of free drainage, following cystotomy. Any urinary obstructions should be relieved. Many cases go on to the stage of contracted bladder with consequent infected ureterohydronephrosis. Occasionally, the contraction of the bladder is so marked that help is obtained only by plastic operations. Schüppel cites a case in which there were multiple urethral strictures.

Decompression.—Bonneau [43] states that in the presence of marked distention of the bladder in patients who have not been catheterized and

42. Schüppel, A.: Ueber cystitis gangraenosa dissecans und die Therapie ihrer Folgen, Arch. klin. Chir. **145**:380, 1927.

43. Bonneau, M. R.: Evacuation lente de la vessie par drainage sus-pubien filiforme avant la cystostomie, Presse méd. **2**:1350, 1927.

whose urine is normal, slow decompression of the bladder is of great advantage; he empties the bladder by means of a lumbar puncture needle of small caliber, introduced into the bladder suprapubically. The bladder is usually completely evacuated in twelve hours. The urinary equilibrium is reestablished, and secondary cystostomy may be carried out without risk to the patient.

Perrier, in discussing Bonneau's paper, states that he also makes a suprapubic capillary puncture in cases of marked distention. He reports a case in which this procedure was carried out on two occasions; the second was necessary on account of too long a delay before proceeding with the cystostomy.

Gerard, in cases of extreme distention, makes a small button-hole incision in the bladder through which he inserts a small catheter which is opened intermittently every other hour, permitting 200 cc. of urine to escape. The catheter is permitted to drain continuously after the fourth day.

[ED. NOTE.—All stab or punch operations through the suprapubic region carry a moderate risk on account of possibility of injury to the peritoneum and urinary extravasation. The same results as those obtained by Bonneau can be carried out by various methods of decompression with a urethral catheter, notably that described by Foulds and Bumpus.]

Tumors.—Drexler and Ginberg [44] state that during the last eight years Joseph has treated more than fifty patients with benign and malignant tumors of the bladder either by chemocoagulation alone or by chemocoagulation combined with electrocoagulation. The technic is as follows: A cystoscope is passed and the bladder washed and filled as for cystoscopic procedure. A ureteral catheter with an open end is inserted through the cystoscope, and from 20 to 30 drops of freshly prepared solution of trichloracetic acid is run over the upper surface of the tumor. The tumor becomes white due to action of the acid. After treatment the bladder is emptied and washed.

Patients with tumors situated in an area of the bladder inaccessible to the cystoscope can be treated with frequent instillations into the bladder of from 40 to 50 cc. of distilled water containing from 20 to 30 drops of trichloracetic acid. Long-standing hematuria, the result of tumors of the bladder, can also be arrested in this manner. After two or three weeks either the tumor entirely disappears and is replaced by scar tissue, or it is reduced in size and is ready for further treatment.

Chemocoagulation is especially indicated in cases of carcinoma of the bladder with foul-smelling necrosis and bleeding with secondary cystitis.

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44. Drexler, L. S., and Ginberg, W.: Treatment of Bladder Tumors by Chemocoagulation, Surg. Gynec. & Obst. **45**:820, 1927.

In this way, the necrosis and bleeding can be cleared up and the tumor reduced in size sufficiently to allow further treatment with electrocoagulation.

According to experiments on animals carried out by Drexler and Ginberg, chemocoagulation apparently does not have any permanent injurious effect on the normal mucosa of the bladder.

Ulceration.—Dean [45] reports three cases of ulceration of the bladder, a late effect of applications of radium to the uterus. These cases are usually seen by urologists, and the symptoms and appearance suggest carcinoma of the bladder, but a tumor is not present.

There is often a history of irradiation with radium to a nearby organ, and there is an area of necrosis at the site nearest the application of radium. A histologic section is at times necessary to rule out a diagnosis of carcinoma or tuberculosis. There is an interval of at least a year between the time of the irradiation and the onset of symptoms.

The distressing subjective symptoms have been relieved readily by a prescription consisting of sodium citrate, 20 Gm., tincture of hyoscyamus, 25 cc., and enough cinnamon water to make 120 cc., given 1 teaspoonful in water two hours after each meal.

The most satisfactory treatment for the lesion consists of injections of mercurochrome-220 soluble into the empty bladder. To prevent undue irritation, the earlier instillations consist of 15 cc. of a 1:200 dilution, administered three times a week. Later, when healing results in greater toleration, the strength of the solution may be increased gradually to 2 per cent, the same quantity being used as before at the same time intervals. It is important that this treatment be carried on with regularity and persistence.

[ED. NOTE.—In some cases, it is almost impossible to distinguish between radium irritation, ulceration or malignancy of the bladder, especially in cases in which radium has been applied for primary tumor of the bladder. The area in the bladder is usually reddened, raised and edematous, and has all the appearances of a primary lesion of the bladder. Another point is that brought out by Dean in this paper that there are sometimes intervals of at least a year between irradiation and the onset of the symptoms.]

Exstrophy.—Colby [46] reports a case of exstrophy of the bladder in a boy, aged 7. At one operation the left ureter was isolated from the bladder and transplanted to the sigmoid. Three weeks later at a second operation, the right ureter was transplanted to the cecum; it was neces-

45. Dean, A. L.: Ulceration of Urinary Bladder as Late Effect of Radium Applications to Uterus, J. A. M. A. **89**:1121 (Oct. 1) 1927.

46. Colby, F. H.: Exstrophy of Bladder; Report of Case, Boston M. & S. J. **96**:1033, 1927.

sary to use the cecum to receive this ureter to avoid tension. Photomicrographs of the tissue removed from the bladder showed numerous mucus-secreting glands.

[ED. NOTE.—The mucus-secreting surface, described by Colby, is common in most cases of exstrophy of the bladder; it is the result either of metaplasia of the normal covering or of hyperplasia of glands in the mucosa. Such glandular structure often shows characteristics of malignancy.]

TESTIS

Torsion.—Ormond [47] states that about 150 cases of torsion of the spermatic cord are on record, the first case having been reported by Delasiuave in 1840. It occurs most often near the age of puberty, although a few cases are reported shortly after birth and a few at 60 years or more. The condition has also been observed at birth. The right side is slightly more affected than the left. Developmental errors most often associated with undescended testes (52 to 69 per cent of cases) have been held to be a predisposing cause.

Etiologic factors mentioned are an abnormally loose scrotum, a voluminous tunica vaginalis, an unusually long gubernaculum, and an abnormal attachment of the cord to the testis. From a consideration of the anatomy, Ormond holds that the last two are the most probable etiologic factors. Ordinarily, the reflection of the parietal layer of the tunica over the epididymis forms a mesentery running from the globus minor up to the middle of the globus major. In most of the recorded cases of torsion, this has been much shortened and includes only the globus major, and the twist occurs in this very much shortened mesentery.

Ormond notes in the literature five cases of torsion in intra-abdominal testis and adds a case of his own, that of a man, aged 42, who had experienced intermittent right-sided abdominal pain for two days before admission. At operation a mass was found consisting of a black swollen testis, with one complete twist of its mesentery, the testis being adherent to the tip of the appendix. There was no evidence of malignancy, which had been noted in three cases in the literature. In only one case (Stiles) was preoperative diagnosis definitely determined.

In all these cases, the testis was on the right side. This is not true of torsion generally, although the right side is more frequently affected than the left, just as it is in maldescent of the testis, which seems to be the chief etiologic factor in torsion.

Ormond hypothesizes that torsion in the intra-abdominal testis may be found usually on the right side because of the presence of the cecum with its intermittent loading and pull on the peritoneum.

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47. Ormond, J. K.: Torsion of an Intra-Abdominal Testis, Ann. Surg. **85**:280, 1927.

[Ed. Note.—Torsion of the cord is so frequently associated with descended or partly descended testes in which some developmental error is present that it is interesting to note the occurrence of the condition in the abdomen. Thus another infrequent type of lesion must be considered in the differential diagnosis of disease of the lower right quadrant. The absence of the testis from the scrotum might point to such a lesion if other features of the condition are not contradictory.]

Tumor.—Roeder [48] describes a case of hyperplasia of the subperitoneal fat beneath the mesothelial coat (tunica vaginalis viscerum) of the testis. The patient had a left indirect inguinal hernia with a hydrocele of the tunica vaginalis. At operation, surrounding the cord and beneath the sac, was a continuous and complete layer of fat extending from the testis up to and through the internal inguinal ring. After the sac of hydrocele had been dissected away, a small nodule beneath the testicular layer of the tunica vaginalis was noted. This proved to be a lipoma, well encapsulated, and more intimately attached to the tunica vaginalis than to the tunica albuginea.

In reviewing the literature, attention is called to the fact that the scrotum embryologically is formed by a constriction of the walls of the lower part of the abdomen. The constriction includes the peritoneum and subperitoneal areolar tissue, the presence of which naturally favors the deposition of fat. Therefore, so-called fatty tumors of the testis should be regarded as hyperplasia of the subperitoneal fat originating from the areolar tissue beneath the tunica vaginalis viscerum or the testicular peritoneum. To be carefully differentiated, are masses of fat along the mesothelial tunics of the cord and fatty tumors in the region of the inguinal canal. Eight cases of lipoma of the testis are reported from the literature and analyzed. In the majority of these the tumor probably originated from the lower end of the cord and therefore cannot be classified as true testicular tumor, by the foregoing criteria.

[Ed. Note.—In differentiating scrotal tumors one seldom thinks of the possibility of lipoma, a lesion which occurs surprisingly at times in other parts of the body. Roeder calls attention to the embryologic occurrence of this tissue in the scrotal contents and the clinical possibility of encountering such cases should be borne in mind.]

Infarction.—Lubash [49] reports a case of infarction in the testis. The testis belongs to the same category as the kidney, spleen and brain, in that its circulation is terminal. Since infarction of the testis exists, it should be thought of as an entity.

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48. Roeder, C. A.: Lipoma of the Testicle, Ann. Surg. **85**:275, 1927.
49. Lubash, Samuel: Infarction of the Testicle, J. Urol. **18**:421, 1927.

The etiology, although still obscure, may be classified as follows: (1) predisposing, any condition that could cause embolism; (2) exciting, a temporary twist of the cord that may result in stasis in one of the arterioles. Infarction of testicle should be suspected in patients with temporary torsion whose symptoms return after abatement. The treatment is purely surgical in the form of orchidectomy.

URETHRA

Chauvin [50] distinguishes four groups of double urethra: 1. The complete double urethras which are more or less permeable canals extending from the bladder to a definite meatus; there is no connection between the two urethras. 2. The one-eyed juxtra-urethral ducts, which end in a culdesac at one or the other of the extremities open either into the bladder or to the exterior. 3. The bifurcation type of unique, and it is the anterior segment which bifurcates into two portions. into two branches of variable calibers. At times the deep urethra is unique, and it is the anterior segment which bifurcates into two portions. In other cases the reverse disposition, in which the anterior is single and the posterior portion is double, is found. The accessory canal in this form may be on top, underneath or on the side of the principal urethra. 4. The fourth type comprises the canaliculized diverticula; they communicate by one of their extremities. In this type are merely small cellules which usually dip down into the spongy or prostatic urethra.

UROLOGIC DIAGNOSIS

Kretschmer [51] points out that all adult patients who present symptoms of lesions in the genito-urinary tract are generally subjected to a complete urologic examination. This is not ordinarily the case with children, although infants and children are subject to almost all the urologic diseases of adult life, except enlargement of the prostate and carcinoma. As a matter of fact, small children can be subjected to a complete urologic examination. There are seven patients in this series under 12 months of age. The youngest was a boy, aged 27 days. Thirty-four of the patients were males and fifty-two were females.

The symptoms in children and adults are about the same in order of incidence: fever, pain, cloudy urine, frequency and hematuria.

In disorders of the kidney, pyelitis leads the list, followed by tuberculosis. The patients with persistent pyelitis were treated by pelvic lavage with solutions of silver nitrate.

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50. Chauvin, E.: À propos des urètres doubles; en particulier de leurs variétés postérieures, J. d'urol. méd. et chir. **23**:289, 1927.

51. Kretschmer, H. L.: Urologic Problems in Infancy and Childhood, J. Urol. **18**:433, 1927.

Stricture of the ureter was seen in ten patients, three boys and seven girls. Diverticula and tumors of the bladder were also encountered.

It is shown that the urinary diseases of childhood resemble those of adult life, and that with practice, cystoscopy in children can be accomplished without difficulty, and may be useful for therapeutic procedure as well as for diagnosis.

Hunter and Montgomery [52] follow up the work of Andrewes, who, when investigating the hypobilirubinemia indicated by the van den Bergh test in uremic serum, discovered a new type of diazo-reaction. On allowing the mixture of alcoholic extract from a uremic serum to stand in contact with the van den Bergh reagent for a period of twenty-four hours, Andrewes found that a pinkish-brown or buff color developed, and that when this was made alkaline with a strong alkali, a bright red color appeared immediately; this was stable for from a few minutes to more than half an hour according to its original intensity.

Hunter and Montgomery observed nephrectomized rabbits and dogs, and thus confirmed the work of Andrewes. As a result of their investigations, they modified the technic previously used and presented the following:

One gram of sulphanilic acid is added to a liter flask containing about 500 cc. of water and 15 cc. of 37 per cent (concentrated) hydrochloric acid. The contents are shaken at ordinary temperature till the sulphanilic acid has gone into solution. Water is then added to the contents mixed. The solution keeps indefinitely.

It is more convenient and reliable to have a stock 5 per cent solution of sodium nitrite than to make 0.5 per cent solutions at weekly intervals. Five grams of good quality sodium nitrite is dissolved in 100 cc. of water. This, if kept in a stoppered bottle in an icebox, is reliable for at least a year, and for several months without the latter precaution. One cubic centimeter of this solution is mixed with 9 cc. of water before each diazotization.

The test is performed by precipitating and centrifugalizing the serum or plasma with 2 volumes of 95 per cent alcohol, taking 1 cc. of the supernatant fluid in a small test tube, adding 0.4 cc. of the diazo-reagent and 0.1 cc. of concentrated hydrochloric acid. The tube is heated over a flame or in a water bath at about 80 C., for a minimum of two minutes. If the test is positive, the buff color will then have developed fully. The tube is cooled and 1 cc. of 2.5 normal solution of sodium hydroxide is added and the tube quickly inverted. The intensity of the red color thus produced accords with the buff color obtained in the acid solution. As the buff color is stable, it is the more suited for

52. Hunter, George, and Montgomery, R. C.: On the Diazo Reaction in Uremic Serum and in Normal Urine, Canad. M. A. J. **17**:148, 1927.

comparative tests. At any rate, it can be preserved until it is convenient to add alkali to a series of tubes at one time.

As originally pointed out by Andrewes, the secondary red color is produced only by the addition of strong alkali. This color will not develop with strong alkali in the presence of a large amount of ammonia, so that ammonium sulphate cannot be used in this test in association with alcohol to precipitate the proteins from the serum, as recommended by Thannhauser and Andersen for the van den Bergh test.

Their technic makes the test more sensitive and renders it less subject to interference. The substance giving the diazo-test in uremic serum is shown to be a normal, but as yet unidentified, urinary constituent.

Gottlieb and Strokoff [53] report several cases in which pneumopyelography was employed. They drew the following conclusions from their study: 1. This is a method of choice on account of the absence of irritation even in cases of hydronephrosis. 2. It permits the obtaining of pictures of stones which are not usually opaque in roentgenograms. 3. Only one roentgenogram, instead of two, is necessary to make the diagnosis. 4. Its technical application is simple and harmless when properly used.

[ED. NOTE.—Pneumopyelography has not been used extensively in this country. Apparently from reports in American periodicals the results are not so successful as reports from foreign clinics seem to indicate. As cystoscopy and pyelography are such satisfactory diagnostic procedures, and in most cases permit a precise diagnosis, pneumopyelography would have to be markedly improved before it is used to any extent in American clinics.]

Marion [54] gives a number of general principles in diagnosis and treatment of urologic conditions. He states that it is necessary to establish the diagnosis of a disease by relying on one physical symptom, such as pyuria or hematuria, rather than on a functional trouble, such as pain or frequent urination.

If, during urethral catheterization, obstruction is encountered in the region of the bulb of the urethra, a Béniqué catheter should be used and not a sound or a bougie of smaller caliber.

Hematuria in a patient with prostatic hypertrophy must not be attributed to the prostatic enlargement unless all other causes of hematuria have been eliminated.

A patient suffering from chronic urinary distention caused by prostatic hypertrophy should not be catheterized; the bladder should be drained and explored.

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53. Gottlieb, J. G., and Strokoff, F. J.: La pneumopyélographie, J. d'urol. méd. et chir. **23**:328, 1927.

54. Marion, G.: Quelques grands principles directeurs du diagnostic et de la thérapeutique urologiques, J. d'urol. méd. et chir. **23**:194, 1927.

All cases of spontaneous cystitis, all cystitis resisting the usual treatment for this condition and recurring cases of cystitis suggest tuberculosis. In all cases of cystitis, except those following urethritis, cystoscopy should be carried out. Catheterization of the urethra should not be preceded by a general examination of the bladder. Any type of hematuria demands immediate urologic examination.

All patients with renal pyuria which is not definitely tuberculous require roentgen-ray examination. All roentgen-ray examination for renal stone should include the entire urinary tract.

The use of vaccines is often a "cache-misère," masking the physician's ignorance and his incapability of establishing a diagnosis of infection. The diagnosis should be made primarily, and vaccine should never be used unless other methods are useless.

Marion,[55] in discussing the value of phenolsulphonphthalein, insists on the following points: 1. One should always use phenolsulphonphthalein of the same origin. 2. In an intravenous injection, it is necessary that the entire amount be injected into the vein. 3. The measure of standard must be frequently renewed. 4. If the result is doubtful, the test must be repeated several times.

A patient eliminating large quantities of phenolsulphonphthalein can usually be considered as having good resistance. In cases in which the amount of phenolsulphonphthalein is found low on several occasions, the surgical outlook is usually poor.

The phenolsulphonphthalein test is more reliable than the so-called Ambard's "constant"; if fever is present, the constant improves, whereas the amount of phenolsulphonphthalein eliminated diminishes, giving a more accurate index of the condition.

Pescatori [56] cites a case which he called "false appendicitis." The patient had pain in the right hypochondrium, radiating into the ileocecal fossa and occasionally to the left side. A diagnosis of chronic appendicitis was made, but roentgenologic examination revealed a "bean-sized" stone in the left kidney. Roentgenograms of the gastro-intestinal tract were negative. Removal of the stone was followed by uneventful recovery. Pescatori calls attention to this case, and it is cited here to show the valuable use of the roentgen ray in differential diagnosis, a point in technic used in American urologic circles for a long time.

[Ed. Note.—This report is of interest in that the clinical course is the opposite to that usually observed. Frequently, patients with urinary calculi are subjected to one or more abdominal operations before the true nature of their condition is determined. In a series of more than

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55. Marion, G.: À propos de l'epreuve de la phénosulfophthaléine, J. d'urol. méd. et chir. **24**:57, 1927.

56. Pescatori, Guido: Sindrome appendicitica da calcolosi renale sinistra rivelatasi ai raggi X, abstr. Ztschr. f. urol. Chir. **21**:13, 1926-1927,

1,400 patients with renal stone, 50 per cent had had at least one operation and 20 per cent, two or more operations before the renal stone was removed. In a second series of 400 cases of ureteral calculi, either the gallbladder or the appendix had been removed in 23 per cent. In at least half of these cases, stones had been present in the left ureter.]

Jeck and Munch [57] point out the truth of the words of Watson, who says: "In the examination of urine for tubercle bacilli, the method which requires the fewer laboratory reagents, consumes less time, and gives a higher percentage of positive results is the one which will be used eventually." They, therefore, carried out a most thorough and convincing group of experiments, proving the value of simple centrifugalization as a method of identifying the bacilli of tuberculosis in the urine. They found that in examining any specimen of urine for the bacilli by centrifugalizing methods, the best results were obtained when the specimen was centrifugalized for a period of not less than fifteen minutes at a speed of approximately 2,300 revolutions a minute. Longer than fifteen minutes is apparently unnecessary.

Pus is important, as a centrifugalizing force, in carrying down the bacilli. According to certain established methods, part of the pus should be removed in order to produce better smears; then the pus removed should be examined for bacilli as well as the final sediment. Coincident centrifugalization of part of the unknown specimen to which bacilli from a stock suspension have been added and subsequent coincident staining of both smears on the same slide are advisable.

Simple centrifugalization as a means of finding the bacilli of tuberculosis in the urine is equal to any established method and better than most of them. It would, therefore, seem to be the most satisfactory method for the urologist who does his own laboratory work or who has it done under his own supervision.

Lower [58] favors the use of the anoci-association method of anesthesia from the standpoint of minimizing traumatic and psychic shock. The lipoid solvent anesthetics, such as ether and chloroform, have a special deleterious effect on the kidneys. Albuminuria follows their usage in about a fourth of the cases. In patients with diseased kidneys, the effect of inhalation anesthesia of this type may be enough to produce fatal urinary suppression or persistent albuminuria. Lower thus believes that the use of ether and chloroform is contraindicated in renal operations or when the urinary tract is already carrying an extra load.

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57. Jeck, H. S., and Munch, Margaret A.: Studies in Centrifugalization as a Means of Identifying Tubercle Bacilli in the Urine, J. Urol. **18**:607, 1927.

58. Lower, W. E.: Anesthesia in Genito-Urinary Operations, Ann. Surg. **86**: 268, 1927.

A choice is given of nitrous oxide, ethylene, spinal anesthesia, sacral block and local infiltration. Spinal anesthesia is disapproved because of its effect on the blood pressure, preference being given to sacral block and local infiltration, for suprapubic and scrotal operations and to regional block, local infiltration and analgesia by nitrous oxide oxygen for operations on the kidneys, at times to be supplemented with local infiltration.

In operations on the bladder and prostate, sacral block, combined with infiltration of the abdominal wall, is recommended, the technic of Hunt being followed. The wall of the bladder is also infiltrated, as well as the capsule of the prostate, if this is the point of attack. Usually this procedure will suffice, but the anesthetist must reassure the patient; if there is apprehension, he should be ready to administer nitrous oxide to the point of analgesia. The patient is for this reason given a preliminary hypodermic injection of morphine and atropine.

In the genito-urinary examining room, Lower prefers the use of procaine to cocaine as a urethral anesthetic, especially in operations such as urethrotomy. For cystoscopy 10 cc. of a 2 to 4 per cent solution of procaine is used. If a pyelogram is to be made, morphine is not given, as Lower believes that the patient should be sensitive to the pain of renal distention. In cases of tuberculosis of the bladder, sacral anesthesia finds a field of usefulness. It is contraindicated in punch operations because of the fact that complete relaxation of the neck of the bladder frequently follows sacral block. Local infiltration of the neck of the bladder, together with analgesia by nitrous oxide, finds a field of usefulness here.

In the case of old men with enlarged prostates, the combination of sacral block and abdominal wall infiltration is emphasized as being a life-saving procedure.

[Ed. Note.—Lower expresses the thought of the more progressive urologists of the day. Sacral block is easily administered in the average case, and with infiltration of the abdominal wall as a rule no further injection of the bladder or prostate itself will be needed. If the anesthesia is properly administered, it may be said also that pain will as a rule be absent, and there will seldom be need for nitrous oxide or any form of anesthesia by inhalation unless it is given because of apprehension or mental confusion of the patient. Lower uses procaine as an anesthetic for the urethral mucosa. It has been the experience of most urologists that procaine has little surface action; in order to have an anesthesia with a satisfactory surface action, it is usually necessary to use cocaine.]

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